JOURNAL
of the
American Veterinary Medical Association

Formerly AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

Vol. LXXXV, N. S. Vol. 38 JULY, 1934 No. 1

THE SWINE SITUATION

Practicing veterinarians throughout the Corn Belt have been reporting that the vaccination of pigs against cholera this year is off from 25 to 50 per cent. Two outstanding reasons account for this falling off in the use of prophylactic vaccination: (1) the low market prices for all classes of hogs and (2) a marked reduction in the number of spring pigs farrowed. In all probability two other closely related factors have contributed to the situation: (1) the financial straits of agriculture in general and (2) the customary willingness of farmers to gamble with chance.

In spite of some predictions to the contrary, a reduction in the number of spring pigs farrowed appears to be an actuality. The U. S. Bureau of Agricultural Economics reports a decrease of 23 per cent in the spring pig crop, as compared with the 1933 farrow. An even greater decrease is predicted for fall pigs. The actual number of pigs on June 1 is reported at 37,427,000 head, a shrink of almost 15,000,000 head from the 1933 figures.

During recent months the newspapers have reported quite a number of unusually large litters of spring pigs in various localities. Predictions have been made that nature would compensate for the hog slaughter of the past fall by providing larger litters for the sows bred for spring farrow. According to the government figures, nature apparently has gone along in the even tenor

of her way, as far as the size of 1934 litters is concerned. As a matter of fact, the average litter this spring appears to be slightly smaller than in 1933—5.83 pigs per litter this year against 5.87 last year.

As would be expected, those states which were most seriously affected by the drouth last year show the greatest shrinkage in the hog population. South Dakota is reported to show a 45 per cent reduction this spring.

Hogs six months of age on June 1, according to the B. A. E. census, were off about 3,700,000 head, with the greatest decrease in the Dakotas and Kansas, and the smallest decrease in Missouri.

Veterinarians throughout the Corn Belt are feeling the effects of the falling off in spring vaccination. Some close observers fear that the stage is being set for a bad outbreak of cholera during the late summer or early fall. A large percentage of non-immune hogs is always a contributing factor to an extensive outbreak of hog cholera. As far as we know, the only soil for the propagation of the virus of hog cholera is to be found in the bodies of susceptible swine. The possibilities are only too apparent to those who know.

Every possible encouragement should be given to the farmer who wants to protect his herd against cholera under present conditions. Even in a year when there is less vaccinating than usual, there is a large amount of hog cholera virus being spread around indiscriminately by those who really should be forbidden to handle virus, in the best interests of the swine industry. As long as the use of virus is unrestricted, just so long will it be necessary for large numbers of swine-owners to protect themselves by having their herds vaccinated each year.

HOUSE OF REPRESENTATIVES

The House of Representatives of the American Veterinary Medical Association will function for the first time during the 71st annual convention in New York in August. The time for the meeting of the new body has been fixed for Wednesday afternoon, August 15, at 4 o'clock.

It would be both timely and in order to point out again just what is hoped to be gained by this departure from previous custom. It may be stated in one sentence. The main object of delegating the work of transacting the strictly business part of our annual conventions to a small, representative group of members is to conserve the time of the rank and file of the members

who attend these meetings. The gradual but steady increase in the activities of the national organization naturally has resulted in increasing the amount of business to be transacted at each annual convention. This has been in spite of the fact that the Executive Board handles many matters that do not come before the Association for action.

The slim attendance at some of the business sessions of recent conventions is the best evidence that the majority of our members are not particularly interested in what might be called the routine business of the Association. There have been business sessions when a quorum was not present. (Article 4, Section 1, of the By-laws provides that 25 per cent of the members registered at a meeting shall constitute a quorum for the transaction of business.)

Voting strength in the House of Representatives, as provided in Section 13 of Article 5 of the Constitution, is determined by the number of A. V. M. A. members in each state on August 1. States having 50 A. V. M. A. members or less will be entitled to one vote; 51 to 150 members, two votes; 151 to 300 members, 3 votes; 301 to 450 members, 4 votes; 451 or more members, 5 votes.

On the foregoing basis, the 40 states having affiliated to date will be entitled to the number of votes shown in the following table:

Alabama	1	Nebraska	2
Arizona	1	Nevada	1
California	4	New Jersey	2
Colorado	1	New York	3
Connecticut	1	North Carolina	1
Delaware	1	North Dakota	1
Florida	1	Ohio	3
Georgia	1	Oklahoma	2
Illinois	3	Oregon	1
Indiana	2	Pennsylvania	3
Kansas	2	Rhode Island	1
Kentucky	1	South Carolina	1
Louisiana	1	South Dakota	2
Maine	1	Tennessee	1
Maryland	2	Texas	2
Massachusetts	2	Utah	1
Michigan	3	Vermont	1
Minnesota	3	Virginia	2
Missouri	2	Washington	1
Montana	1	Wisconsin	2

The above calculation is based on the membership as shown by the report of the Secretary of the A. V. M. A., one year ago, and in a few cases the number of votes may vary from the figures shown, as a result of membership changes (delinquencies, resignations, removals, deaths, etc.) during the past year.

EXECUTIVE BOARD ELECTIONS

The Executive Board elections which came to a close during the past month resulted in the re-election of both incumbents. In District 4 (Kentucky, West Virginia, Virginia, Maryland, District of Columbia, Tennessee, North Carolina, South Carolina, Georgia, Alabama, Mississippi, Florida, Cuba, West Indies, South America) Dr. C. A. Cary, of Auburn, Ala., was elected for another five-year term, beginning at the close of the meeting in New York in August. In District 10 (Ohio and Michigan) Dr. O. V. Brumley, of Columbus, Ohio, who has served his District since it was organized in 1930, was returned an easy winner over the other candidates. His new term also will begin at the close of the New York meeting, and will expire in 1939.

APPLICATIONS FOR MEMBERSHIP

In spite of the fact that the membership campaign in behalf of the Twelfth International Veterinary Congress has occupied the center of the stage during recent months, quite a few applications for membership in the A. V. M. A. have been received during the past thirty days, as will be seen by the list of new applications given first listing this month. This is the largest list published in nine months.

It has been our custom, for some time, to publish, twice a year, Section 1 of Article 2 of the By-laws, explaining the *modus operandi* for filing an application for membership in the A. V. M. A. The custom is being continued this month, as we have found that it saves considerable letter-writing. Here is how it is done:

> Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possble, said application, with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BADER, MADERO N. 1106 Avenue K, Galveston, Texas
D. V. M., Agricultural and Mechanical College of Texas, 1932
Vouchers: Frank Hecker and H. V. Cardona.

CLARK, E. M. 4120 S. 20th St., Omaha, Nebr.
D. V. M., Saint Joseph Veterinary College, 1915
Vouchers: Harvey E. Smith and W. C. Herrold.

DANNER, HOWARD 1725 S. Meridian St., Indianapolis, Ind.
D. V. M., Indiana Veterinary College, 1906
Vouchers: D. W. Gerber and R. A. Craig.

ELMER, SAM New Glarus, Wis.
D. V. M., Ohio State University, 1934
Vouchers: W. F. Guard and O. V. Brumley.

FRANZ, G. A. 4321 Emmet St., Omaha, Nebr.
D. V. M., Kansas State College, 1918
Vouchers: Harvey E. Smith and W. C. Herrold.

HAMANN, E. E.. 2601 E. Lansing Ave., Lansing, Mich.
D. V. M., Michigan State College, 1931
Vouchers: I. Forest Huddleson and D. Blaire Meyer.

JONES, EUGENE C. 9088 Santa Monica Blvd., West Hollywood, Calif.
B. S., D. V. M., State College of Washington, 1924
Vouchers: W. L. Curtis and C. A. White.

KERMEN, WM. R. 610 E. Third St., Cle Elum, Wash.
B. S., D. V. M., State College of Washington, 1934
Vouchers: E. E. Wegner and Wm. J. Pistor.

LANG, EDWARD M., JR. 716 E. Broadway, Louisville, Ky.
D. V. M., Ohio State University, 1934
Vouchers: W. F. Guard and O. V. Brumley.

LIROCCHI, VINCENT W. 2820 S. Claiborne Ave., New Orleans, La.
D. V. M., Ohio State University, 1934
Vouchers: W. F. Guard and O. V. Brumley.

MILLER, LUTHER P. 114 E. Maple St., Clyde, Ohio
D. V. M., Ohio State University, 1934
Vouchers: W. F. Guard and O. V. Brumley.

PATTERSON, E. E. 3800 Grand River Ave., Detroit, Mich.
D. V. S., Grand Rapids Veterinary College, 1901
Vouchers: F. M. Blatchford and B. J. Killham.

PATTERSON, JAS. E. 14411 Saint Marys Ave., Detroit, Mich.
B. V. Sc., Ontario Veterinary College, 1925.
Vouchers: F. M. Blatchford and B. J. Killham.

PIERCY, PAUL L. University of Missouri, Columbia, Mo.
D. V. M., Iowa State College, 1933
Vouchers: O. S. Crisler and Cecil Elder.

WINSLADE, W. A. Shipman, Ill.
D. V. M., Iowa State College, 1934
Vouchers: C. H. Stange and C. H. Covault.

Applications Pending

(See June, 1934, JOURNAL)

SECOND LISTING

Barber, P. G., 291 Main St., Wakefield, R. I.
Gates, Cecil L., 10 Washington Ave., Belleville, N. J.
Martin, F. E., 610 S. High St., West Chester, Pa.
Morin, J. R., Windsor Ave., Rockville, Conn.

COMING VETERINARY MEETINGS

Minnesota State Veterinary Medical Society and University of Minnesota Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 5-6, 1934. Dr. C. P. Fitch, Secretary University Farm, Saint Paul, Minn.

Wisconsin Veterinary Medical Association. Appleton, Wis. July 9-10, 1934. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Northwest Veterinary Medical Association. (Joint meeting of the Oregon, Washington and British Columbia Veterinary Medical Associations.) Salem, Oregon. July 9-11, 1934. Dr. B. T. Simms, Secretary, Oregon State Agricultural College, Corvallis, Ore.

North Carolina State Veterinary Medical Association. Joint meeting with South Carolina Association of Veterinarians. Spartanburg, S. C. July 10-11, 1934. Dr. J. Howard Brown, Secretary, Tarboro, N. C.

South Carolina Association of Veterinarians. Joint meeting with North Carolina State Veterinary Medical Association. Spartanburg, S. C. July 10-11, 1934. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Maine Veterinary Medical Association. Gateway Inn, Portland, Maine. July 11, 1934. Dr. R. E. Libby, Secretary, Richmond, Maine.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 11-12, 1934. Dr. E. A. Caslick, Secretary, Paris, Ky.

Tulsa County Veterinary Association. Tulsa, Okla. July 12, 1934. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Western New York Veterinary Medical Association. Letchnorth Park, near Portageville, N. Y. July 12, 1934. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

New Jersey, Veterinary Medical Association of. Hotel Monterey, Asbury Park, N. J. July 12-13, 1934. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Virginia State Veterinary Medical Association. Orange, Va. July 12-13, 1934. Dr. I. D. Wilson, Secretary, Virginia Polytechnic Institute, Blacksburg, Va.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. July 17, 1934. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Missouri Veterinary Medical Association. Missouri Hotel, Jefferson City, Mo. July 17-18, 1934. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

National Veterinary Medical Association of Great Britain and Ireland. Edinburgh, Scotland. July 30-Aug. 3, 1934. F. Knight, Esq., General Secretary, 2, Verulam Buildings, Gray's Inn, London, W. C. 1, England.

Northwestern Ohio Veterinary Medical Association. Bowling Green, Ohio. August 2, 1934. Dr. Warren P. S. Hall, Secretary, Division of Health, 9 Ontario St., Toledo, Ohio.

Poultry Science Association. A. & M. College of Texas, College Station, Texas. August 7-10, 1934. Prof. D. H. Reid, President, A. & M. College of Texas, College Station, Texas.

Twelfth International Veterinary Congress. Waldorf-Astoria Hotel, New York, N. Y. August 13-18, 1934. Dr. H. Preston Hoskins, General Secretary, 221 N. La Salle St., Chicago, Ill.

American Veterinary Medical Association. Waldorf-Astoria Hotel, New York, N. Y. August 14-16, 1934. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

Civil Service Examination

The U. S. Civil Service Commission announces an open competitive examination for "Associate Veterinarian (Diseases Affecting Wild Animal Life)." Applications for the position must be on file with the Commission not later than July 23, 1934.

Competitors will not be required to report for a written examination, but will be rated on their education and experience and on a thesis or published writings. A requirement is not less than three years of responsible professional experience, at least one year of which must have been in the treatment of, or research in, the diseases of native wild animal life. Ordinary veterinary practice will not be accepted as complying with the requirement of the one year of experience in the diseases of wild animal life. The entrance salary is $3,200 a year, subject to a deduction not to exceed 5 per cent during the fiscal year ending June 30, 1935, as a measure of economy, and also to a deduction of 3½ per cent toward a retirement annuity.

Full information may be obtained from the Secretary of the U. S. Civil Service Board of Examiners at the post office or customhouse in any city, or from the U. S. Civil Service Commission, Washington, D. C.

Bird's Eye View of Program

MONDAY AUG. 13	TUESDAY AUG. 14	WEDNESDAY AUG. 15
9 A. M. Meeting of Permanent Commission	9 A. M. General Session of Congress Meeting of Women's Auxiliary	9 A. M. General Session of Congress
10:30 A. M. Opening Session of Congress (Ladies attend)	2 P. M. Sectional Meetings of Congress Sightseeing trip for ladies	2 P. M. Sectional Meetings of Congress Trip to Radio City Music Hall for ladies
12 NOON Luncheon for ladies. Fashion show and tea	6 P. M. Alumni Meetings	4 P. M. A. V. M. A. House of Representatives
2 P. M. Sectional Meetings of Congress	8 P. M. Opening Session of A. V. M. A. Convention	7:30 P. M. Official Banquet
8:30 P. M. Reception		

for Week of August 13-18, 1934

THURSDAY AUG. 16	FRIDAY AUG. 17	SATURDAY AUG. 18
9 A. M. General Session of Congress	9 A. M. Sectional Meetings of Congress	9 A. M. to 5 P. M. Post-Congress Clinic
——	——	Boatride around Manhattan Island for ladies
2 P. M. Sectional Meetings of Congress	1:30 P. M. Automobile trip to Walker-Gordon Farm and Rockefeller Institute for gentlemen	——
Ladies tour Rockefeller Center and NBC studios	Sightseeing trip for ladies	10 A. M. Meeting of Permanent Commission
——	——	——
4 P. M. Closing Session of A. V. M. A. Convention	——	11 A. M Closing Session of Congress
——	8 P. M. Meeting of Committee on Budapest Prize	——
7 P. M. Congress Banquet		12 NOON Meeting of Permanent Commission

HISTORY OF VETERINARY MEDICINE IN THE UNITED STATES*

By JOSEPH M. ARBURUA, *San Francisco, Calif.*

Introduction

The chronicling of historical data seems to be a phase of veterinary literature that has been sadly neglected in this country. It can be said, we believe without fear of contradiction, that in no other profession can one be found that knows so little of the history and traditions of his profession as the average veterinarian.

An effort to reconstruct the story of the progress and achievements of veterinary medicine in the United States has been a very difficult task and it is apparent to us that the work should be undertaken by one living on the eastern coast of the United States, where our oldest libraries and traditions are to be found.

It is a singular fact, worthy of comment, that some of our colleagues of another day, say fifty or seventy-five years ago, did not undertake this subject when first-hand information was obtainable, and in this way hand down to future generations much information that has been irretrievably lost to posterity.

In examining the vast amount of veterinary literature, at least that which was written prior to 1890, only a few works on American veterinary history are to be found and of these only one can be said to treat the subject seriously. In the January, 1877, number of the *American Veterinary Review* (later the JOURNAL of the American Veterinary Medical Association), which incidentally was the first issue of that journal, we find that the first article[1] is written by Professor Alexander Liautard and titled, "The History of Veterinary Medicine." This treatise covers nineteen pages of fine print and is the most complete work to be found on the subject. It shows that a great deal of work was put into it.

In 1884, at the opening of the veterinary department of the University of Pennsylvania, Professor Rush Shippen Huidekoper, who was at the head of that department, gave an address[2] that was later published in the *American Veterinary Review* and in the *Journal of Comparative Medicine*. This can well be called a synoptical history of veterinary medicine. About two-thirds of it was devoted to ancient history and about one-third to American history. However, on close examination, it is indubitably evident

*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

that Professor Huidekoper had Liautard's article before him when he wrote it.

Huidekoper again, in 1889, when president of the United States Veterinary Medical Association (now the American Veterinary Medical Association), gave an address[3] reviewing the progress of that body since its organization in 1863. This address was published in the *Journal of Comparative Medicine and Surgery*.

Robert Jennings, himself a maker of history, wrote many short articles, for the various veterinary journals, some of them appearing as early as the fifties in the American Veterinary Journal (the first veterinary journal in this country). In most of these articles were written many historical facts which would have been lost to us forever had not he written, but they related only to the activities of his time.

In 1913, when the American Veterinary Medical Association met at the Astor House, New York, that body celebrated the Golden Jubilee of its organization. As most institutions, organizations and individuals are wont to become on their anniversaries, the A. V. M. A. became reminiscent, and one of the outstanding papers presented during that meeting was one sent by Professor Liautard, who was then retired and living in his native France. He had planned attending in person but ill health prevented him from doing so. His paper reviewed the history of the A. V. M. A. as Huidekoper had done previously. The commemoration of the event, by the very nature of the program, awakened in the American veterinarian a realization of the fact that there was no written history of the profession and the result was that a committee on history was created, which has functioned ever since, gathering data with a view of ultimately producing a complete history of our profession in America.

In 1924, Dr. U. G. Houck presented the profession with a very complete work[4] entitled, "The Bureau of Animal Industry of the United States Department of Agriculture," and in the *Veterinary Bulletin* (United States Army), Captain Horace S. Eakins, V. C., U. S. A., has given a detailed history of the United States Army Veterinary Corps from the beginning.[5]

In writing the foregoing it is appreciated by the writer, perhaps better than anybody else, that it is a very feeble and to say the least, an incomplete attempt to record what may be called the history of our profession. But if the reading of what is to follow will serve to excite an interest in the subject and prompt others to take an active part in ferreting out that portion which is missing, and thereby eventually produce a history worthy of

the name, the efforts, time and money expended in this treatise will be handsomely repaid.

The Period Prior to 1800

The history of the veterinary profession in the United States up to the year 1800 can be written very briefly for the reason that, with one exception, no records of any description are available to show in any way the activities of men who applied themselves to the care and treatment of animals.

It must be remembered that the first veterinary college was founded in Lyons, France, in 1761. Consequently, prior to that time, the college veterinarian was non-existent. From that date on, colleges sprang up here and there in most of the capitals of Europe and during the following thirty years some ten such institutions made their appearance. None, however, existed in an English-speaking country until the London College was started in 1791, just nine years before the dawn of the nineteenth century.

It is absurd, of course, to think that any of these graduates would seek an outpost of civilization such as the newly organized republic in North America, when a scarcity of veterinarians existed at home. They naturally were absorbed in their respective countries. Even in Britain, as late as 1801, there were only 37 graduated, qualified veterinarians and the only excuse any of these men could have had for coming to our shores would have been the call of adventure. Even so, records do not show any to have migrated to this country.

The earliest colonists brought with them horses, cattle and other domestic animals, and naturally imposed upon themselves the responsibility of caring for them. It is quite natural to suppose that in each group of colonists was to be found one man who, more than the others, knew or at least purported to know the ailments of animals and their treatment. Perhaps at first this individual imparted his services gratis, in a neighborly spirit, but as he became more proficient and his services more in demand, doubtless he devoted more time to his new profession and demanded remuneration commensurate with his services. Thus it must be realized that from the beginning of the colonization of this country we had with us men who were farriers or cowleeches, practicing this line of human endeavor either as a vocation or an avocation.

The original colonization of the Atlantic coast was, with the exception of New York and northern Pennsylvania, entirely from British stock, and even that territory passed under British con-

trol as early as 1664, after which time Dutch immigration practically ceased and it, in turn, came under British influence. The customs and culture, insofar as local conditions permitted, therefore were British.

Although far behind continental Europe in development, there existed in the motherland a form of veterinary art which was naturally transplanted to this country. This system was comprised of two kinds of animal doctors—the farrier, who treated horses, and the cowleech, who treated cows and other forms of live stock. The former considered himself far superior to the latter. However, one was about as ignorant as the other.

These men of the eighteenth century were of the lowest social caste and their ignorance was appalling. So much so that it was continually being decried by the writers of their time. The source of all information for young America was the motherland and, the protege being less worthy than the tutor, it follows that the same order of things prevailed in this country but to a lesser degree of proficiency, if such a state can be imagined to have existed.

The only source of information open to these men in America, apart from what they inherited from their predecessors, often their fathers, who were as ignorant as themselves, was the current veterinary literature of England, written by such recognized authors as Gibbons, Bartlet, Osmer, James Clark and by others whose products were plagiarisms of preceding plagiarists of Markham's type. No doubt a few consulted these fountains (?) of information, but books of any nature were few, expensive and hard to obtain in early colonial days, as they had to come from across the ocean.

As for local productions along these lines, printing in this country was in its infancy. Thus it is safe to say that American veterinary literature did not appear in this country until the nineteenth century. No evidence to the contrary has as yet been uncovered, or at least it is not common knowledge. It can well be imagined, therefore, that few of the American farriers consulted books.

Nevertheless, we should not be too critical of our colleagues of that day, and we should not attempt to judge them from the present standards of veterinary education. It might be well to consider for a moment the educational qualifications of the practitioners of human medicine. At the beginning of the Revolutionary War, we are informed that there were more than 3,500 physicians within the colonies, of whom not over 400 held degrees from regular medical colleges. Of these latter, practically all

held degrees from foreign schools, since it was only a matter of ten years before the war that the first regular medical college had been established. The Philadelphia college was founded in 1749, and in 1765 a medical department was added to it. The first man to receive a medical degree in this country is thought to be Dr. Archer, who completed the regular course in that school and was graduated in 1768. From this it will be seen that the course was one of three years.

Since colleges were not available and as only a few individuals were in such financial circumstances to be enabled to go to Europe to qualify through a regular collegiate course of instruction, the custom was that of apprenticeship. A young man hired himself to one of the leading surgeons at a very meager salary, if any, and in many instances paid his employer for the privilege of attending him. Usually about four years of apprenticeship were required after which he was given a certificate attesting to his qualifications. This form of apprenticeship was called the preceptor system and the student was said to "ride" with a physician.

History shows us that in all lines of endeavor progress has been enhanced by necessity. All misfortunes are a blessing in disguise. So also the progress of medicine, human and comparative, has shown rapid strides in the face of epidemics and epizoötics. Thus we find that the direct cause of the establishment of the first veterinary college, by Bourgelat, in France, was due to dreadful losses of cattle in that country, showing the need of study along these lines. So likewise the epizoötics in cattle in England and other countries in Europe aroused public interest in veterinary medicine. This natural stimulation of the human mind to combat the invasion of antagonistic influences has always served as a stimulus but seems to be absent in the pioneer days of American existence.

The settlements were sparse and animals were scarce, living closer to nature than is possible in thickly settled regions. This prevented epizoötics of any magnitude, consequently problems of animal pathology did not receive the study of other and older congested countries and progress was limited accordingly.

It is true that there was an outbreak of rabies in Boston in 1785 and again in 1789, regarding which we are informed by Webster that "canine madness" began to rage in all parts of the northern states. "The gazettes of 1785 abound with accounts of this dreadful disease." However, it did not occur to anyone at that time that its control was in anyway related to the veterinary

profession. It was a medical problem handled purely from a human standpoint.

One problem, however, seems to have engaged the attention of the people, particularly those of the southern states. Texas fever, it is generally believed, was introduced into this country by cattle that were imported from the Spanish colonies of Mexico and the West Indies. It is not known when the disease first appeared here but it is definitely known that such cattle had been imported as early as 1610. Though little was known of this disease in the northern states until the last half of the nineteenth century, it must have been a problem in the South, as evidenced by the passage of the following law, in 1795, by the North Carolina State Legislature:

> No person shall hereafter drive any cattle from these parts of this state, where the soil is sandy and the natural production or growth of timber is the longleaf pine, into or through any of the highland parts of the State where the soil or growth of timber is of a different kind, between the first day of April and the first day of November in every year, under the penalty of four dollars for each and every head of cattle so driven to be recovered and applied as before mentioned.

This seems to be the first legislative act passed in connection with animal diseases in these United States and there appears no doubt that it applies to Texas fever.

Apparently the first serious attempt to study this malady by a medical man was by Dr. James Mease, a physician, who was at the time lazaretto (quarantine) physician in State Island, Pa. In August, 1796, he was taking a trip for his health and stopped at Anderson's Ferry in Lancaster County, Pa. He found that his hosts were greatly excited over the loss of several head of cattle while others were sick. Becoming interested, he was informed that a passing herd of cattle coming from South Carolina had occupied, over night, a plowed field belonging to Mr. Anderson. Here several head had been disposed of by sale and all who had introduced the newly purchased cattle into their herds had sustained losses as well as Mr. Anderson. The baffling feature was that none of the transient cattle had manifested any untoward symptoms. This herd stopped a day or two near Downingtown, 32 miles from Philadelphia, where they occupied a field. Sometime later, this field was used by a herd of 260 head of cattle coming from Maryland. Part of the latter herd was sold in this vicinity. They soon commenced to die, and at the same time infected the herds into which they had been introduced. The South Carolina herd was gradually disposed of as it traveled northward, spreading the disease as it went. Dr. Mease interested himself in the matter, performing several autopsies and

prescribing for the sick. He continued to devote much time and study to this malady for upwards of thirty years. Though his first observations were made in 1796, he did not make them public until 1814, when he made his first report to the Philadelphia Society for Promoting Agriculture. This will be referred to again later.

One naturally would wonder how animals were cared for in the army during the Revolutionary War. In European armies farriers and veterinarians had been employed as far back as the time of Augustus Caesar, in the first century, and were considered as indispensable by all civilized peoples. In our army, however, during the War of Independence, written evidence either in general orders or otherwise is yet to be found that men were employed for this purpose.

Mounted troops were not authorized in our military service until March 5, 1792, at which time an act of Congress was passed providing for a squadron of light dragoons composed of four troops. In the tables of organization as authorized, we find that it called for one farrier to each troop. We do not know just what the duties of the farriers were but we can safely assume that among other duties (shoeing, etc.) the care and treatment of animal must have been within their province.

Reviewing the preceding pages, we find that the veterinary art in this country, at the beginning of the nineteenth century, was at a very low state and that the general public had practically ignored the craft as if it was of no economic importance. We do note, however, that the presence of Texas fever was beginning to be felt and, as it is natural to suppose, would ultimately cause concern, causing thinking men to turn to that body of men best equipped to handle the situation. We can see also the faintest signs of a future army veterinary corps being established.

The Period 1800 to 1850

During the period 1800 to 1850, the progress in veterinary medicine was slow, particularly in the first quarter of the century, although periodically some slight interest and activity were manifested. In the 25 years preceding 1850, a definite tendency toward the organization of veterinary medicine along professional lines can be seen, although the calling had not, as yet, gained favorable public approbation.

In presenting this period to the reader, we have been obliged to sacrifice strict chronology in order to avoid confusion, and therefore, have presented the various phases, rather than the individual facts, in chronological order.

Education

Probably the first instances showing that men of consequence were becoming aware of the importance of veterinary science as an economic factor was demonstrated by the activities of Dr. Benjamin Rush in the year 1806. Dr. Rush had been traveling abroad and while there he had had occasion to visit and observe the progress made in the European veterinary colleges by which he was most favorably impressed. Upon his return to America, he addressed a letter to the Agricultural Society of Philadelphia, in which he called attention to the agricultural prospects of this country, and he urged the importance of having a veterinary department established by the University of Pennsylvania. The following year, this letter was discussed before a meeting of the Society, but nothing was done regarding the suggestion of Dr. Rush.

When one considers the professional and political standing of Dr. Rush, his attitude toward veterinary medicine must be considered of importance. It will be well to state that he was one of the outstanding American physicians of his time, having lived from 1745 to 1813. He had the distinction of being one of the signers of the Declaration of Independence, and besides being a professor at the Philadelphia Medical College he was also the founder of the Philadelphia Dispensary, the first one in the United States. He founded also the College of Physicians and Surgeons in the City of Brotherly Love. Early in the year 1777, he was appointed Surgeon General and later in the same year Surgeon-Physician General of the Continental Army. He was the author of several books and served as treasurer of the United States mint from 1799 until his demise, in 1813. Two sons of Dr. Rush likewise became famous. James (1786-1869) was a noted American physician and philanthropist and Richard (1780-1859) was an American statesman and diplomat.

The suggestion of Dr. Rush regarding the establishment of a veterinary college is apparently the only effort in that direction for more than forty years. Toward the end of this period in the history of veterinary medicine, the thought was being entertained, and efforts were being made toward this end, but as they did not assume definite form until much later they will not be discussed in this treatise until a subsequent period.

Infectious Diseases

We have already made reference to Dr. James Mease and his investigations relative to Texas fever as far back as the year

1796. It was not until November 3, 1814, however, that he delivered his first lecture before the Philadelphia Society for Promoting Agriculture, and this, as far as is known, is the first written account of this malady. In that lecture Dr. Mease told that cattle from certain districts in Southern Carolina infected all others with which they came in contact in their travels northward, and that Virginia had prohibited the passage of these cattle through that state.

He was emphatic in his statement concerning the ability of these cattle to spread the disease without their showing the slightest symptoms of the dreaded plague. In fact this peculiarity seemed to baffle and intrigue the doctor. He brought out the fact that cattle imported from Europe, or those brought from the interior to the vicinity of the sea, succumbed to the malady.

On September 20, 1825, he again appeared before the same society and gave an "account of a contagious disease propagated by a drove of southern cattle in apparently perfect health." It was at this time that he gave light to his experiences of 1796, already related.

It was definitely known that cattle from certain districts in South Carolina, as already mentioned, were the carriers but the exact areas from whence these cattle came were not known to Dr. Mease albeit he had endeavored to locate them. It was generally believed that the country of the long-leaf pine was the place where the infection was indigenous but he had been unable to prove this definitely.

In 1836, the North Carolina legislature enacted a law prohibiting the entry of cattle during the months from April 1 to November 1, inclusive, from the states of South Carolina and Georgia. From this year (1836) on, several of the states passed laws of a similar character, or modified existing laws, until eventually a solution was found.

Hog cholera was known in America as early as 1833, at which time it is said to have made its appearance in Ohio. It was here that it was given its descriptive name. Nothing was done regarding the control of this disease until Drs. George Sutton and E. M. Snow, two physicians, devoted considerable time and study to it between the years 1858 and 1862, but no definite results, either in determining its pathogenesis or controlling its spread, were attained until many years later.

In 1843, another scourge appeared in our midst. This time it was contagious pleuro-pneumonia of cattle, frequently called "lung plague." In that year, a cow that had been imported from

Germany was unloaded from the ship to a cowshed in Brooklyn, New York. From this remote source, Long Island cattle became infected and the disease continued to exist there for upwards of forty years before it was controlled.

Before the end of the period under discussion, two more introductions of disease into this country took place. A Mr. Thomas Richardson, of New Jersey, in the year 1847, imported a herd of cattle from England, said to have been valued at $10,000. Symptoms of the disease appeared and, rather than permit the spread of the infection, Mr. Richardson magnanimously slaughtered the entire herd. In 1850, again the disease was introduced through the medium of a cow imported from England, this time again into Brooklyn, where the disease was first discovered in this country in 1843.

It seems rather strange that, despite the fact that this disease was known to exist and could have readily been eradicated if localized to a comparatively small area, nothing was done at the time, and as a consequence in the following decades the penalty for this folly was exacted, and dearly paid, in the tremendous losses resulting from its spread over the entire Atlantic seaboard.

On first thought it is a far cry from the routine functions of the United States Patent Office to veterinary activities, but, nevertheless, through gradual steps it was the Patent Office that was responsible for the establishment of the United States Department of Agriculture, which, in its turn, developed the Bureau of Animal Industry, recognized as one of the outstanding accomplishments in veterinary medicine today, not only in the United States but throughout the entire civilized world.

It is not the purpose of this treatise to give a complete history of the Bureau of Animal Industry, except insofar as it interests us, as veterinarians, in showing the history of the progress of our profession in this country. Anyone desirous of reading a detailed report of the development of this service is referred to the splendid work of Dr. U. G. Houck.[4] To him we acknowledge our indebtedness for the data given herein concerning the Bureau.

In 1837, Dr. Henry L. Elsworth, then Commissioner of Patents, in his report suggested extending aid to farmers by giving advice on agricultural matters and by distributing to them selected seeds for planting. He had been prompted to make these suggestions because in dealing with patent grants he came in contact with farming implements and machinery which were rapidly being improved. This in turn brought him into relations with agricultural interests, including, naturally, animal husbandry. As can

readily be understood, his office became, more or less, a clearing-house for ideas. His advice and suggestions were sought frequently and, as time went on, this office found itself rendering valuable aid to agricultural and live stock interests.

On January 22, 1839, Dr. Elsworth wrote a letter to the chairman of the Commission of Patents in which he penned in part:

> In the discharge of official duties I could not fail to notice facts deeply connected with the subject of agriculture, and so far as I was able, without the neglect of primary obligations, to give all the advantageous aid in my power to this important branch of national industry.

This praiseworthy attitude of Dr. Elsworth resulted in more and more attention being devoted to agriculture and live stock, finally resulting, through successive steps, in our present efficient veterinary body. Thus the foundation was laid in the period under discussion and due credit must be accorded to Dr. Elsworth.

Army

As in the case of the Bureau of Animal Industry, a detailed history of the United States Army Veterinary Corps is not to be gone into, but only as much of it as is necessary to the scope of this treatise will be given in order to bring out the historical picture of the profession as a whole. Again we respectfully refer the reader to other sources, and in this instance we suggest the work of Captain Horace S. Eakins,[5] previously mentioned. It is to this source that we are indebted for our information.

We have previously noted the organization of the first U. S. Cavalry unit, in 1792, in which a farrier was prescribed for each cavalry troop. Beyond this no further reference was made by the War Department, or by Congress, concerning animal care in the armed forces of the country until after the War of 1812, which ended in 1814.

The mounted troops which had been organized had been increased and decreased numerically according to the exigencies of the service from 1792 to 1814, but in the latter year they were reduced to one regiment of cavalry to which was assigned "one principal farrier." The following year, however, the mounted troops were entirely disbanded.

We find no further reference to this matter in official sources until the year 1835, when army regulations required inspectors to see that "veterinarians" perform their duties. This is the first time the War Department had ever employed the word "veterinarian." As there were no veterinarians in the army at that time, it is assumed that this statement must have referred to the farriers. It is, however, possible that veterinarians may have

been hired or retained under a contingent fund, but records available do not give us any information in this regard.

On August 14, 1848, an act of Congress providing appropriations for the support of the army for the fiscal year 1848-49 was passed, in which, under the heading of incidental expenses of the Quartermaster Department, the "hire of veterinary surgeons" and "medicines for horses and mules" were authorized.

From the foregoing we see that the seed that was eventually to grow into our present small, but efficient, Army Veterinary Corps, was planted in the first half of the nineteenth century, but unfortunately it was sowed in poor soil and badly nourished through the years that followed up to the period of the World War.

LITERATURE

American veterinary literature did not make its appearance, that is notably so, until the nineteenth century, and during the first quarter few works came to light. The first that we have been able to trace are articles that appeared in medical journals.

The first of these is to be found in the Medical Repository (New York) in the year 1800. The article is entitled, "Observations on the disease commonly called yellow water in horses," and was written by F. B. Sayre.[6]

In 1805, in a medical journal published in Philadelphia, came to light an article by Barton,[7] on "Some account of the disease called the hollow horn."

In 1808, in the *Philadelphia Medical Museum*, J. Stevenson[8] wrote on "Case of successful treatment of yellow water."

A year later, J. Smith[9] gave an "Account of a disease among neat cattle apparently caused by browsing the juicy sprouts of oak shrubs, in the woods of Suffolk County, New York," in the *New York Medical Repository.*

Before the last two articles mentioned were printed, however, what we believe at this time, for lack of other evidence, to be the very first book that touched upon animal diseases made its appearance. In 1806, Paul Jewett,[10] a farrier of Rawley, Massachusetts, wrote, a small, vest-pocket edition of 67 pages, to which was given the title, "The New England Farrier." The title page, in addition to the title, reads: "in Four Parts: Wherein most of the Diseases to which Horses, Neat Cattle, Sheep and Swine are incident, are treated of; with Medical and Surgical observations thereon." He further tells us that the book is intended for the use of "Private Gentlemen and Farmers," and is "the first production of its kind in New England." It was printed in Hudson, by A. Stoddard.

We are informed in the introduction that the book owes its causes to:

1. The great opportunity I had whilst young of reading authors on farriery, and thereby gained an extensive theory.
2. The extensive practice I have had in this kind of business since, and the reasons experience hath given me to differ from most of the European theories, and confine my observations to practice only.
3. The solicitations of my acquaintances.

From the above we understand that Paul Jewett was not a graduate veterinarian and that he enjoyed a large practice. The third reason is one that most veterinary writers preceding him for the past three centuries have used as an excuse to write on the subject. It is a stereotyped phrase with many of them.

From internal evidence we judge him to have had some education, as he writes clearly, but that he was not abreast of the times although this fault is pardonable when it is realized that all veterinary literature had to come from Europe and, besides being hard to obtain, was of necessity somewhat belated by the time it reached this continent.

In the first part of his book he deals with horses, while the second is devoted to cattle. The third treats of sheep and the fourth of swine. An examination of this book is extremely interesting and, it is believed, shows the practices and knowledge among the best talent in the veterinary profession in our country, during the opening years of the nineteenth century.

The next that interests us was not strictly a veterinary work but rather one of animal husbandry, containing diseases and their treatment, as was the custom of most of the earlier books published in Europe prior to the seventeenth century. The work[11] to which we refer was written by R. R. Livingstone, LL.D., of New York, titled, "An Essay on Sheep." This book was first printed in London, in 1809. The following year, however, it was published in New York, to be followed by another edition in London, in 1811. The work treats of sheep and their diseases in the "American States." It was originally a treatise presented by Livingstone to the New York State Senate, in 1809. The author was the first man to show that merino sheep were capable of thriving in the United States. William Corbett, in 1811, reprinted Livingstone's book and in his (Corbett's) preface he waxed enthusiastic over the fact that henceforth America would not have to depend upon England for clothing. Livingstone's book is well written in splendid English and is very practical as far as the rearing and handling of sheep is concerned, but the portion devoted to diseases was taken from the writings of older English authors and shows nothing new or original.

At this point it may be of interest to note that the first importation of merinos into this country was made in 1802, by Chancellor Livingston, then minister to France.

In 1812, William Taplin's book,[12] several editions of which had appeared in London during the preceding twenty years, was published in Philadelphia. This was a new, combined edition of two volumes in one entitled, "The Gentlemen's Stable Directory; or modern system of Farriery, comprehending all the most valuable prescriptions and approved remedies, accurately proportioned, properly adapted to every known disease to which the horse is incident; interposed with occasional references to the dangerous and almost obsolete practice of Gibson, Brachen, Bartlett, Osmer, and others, etc."

This is probably the first American edition of a British book. Taplin was a surgeon in England who had given up his practice as such and turned to veterinary medicine during the last quarter of the eighteenth century. He was the author of several books on the subject, the above being one of them, his object in writing these books being to advertise himself and his drugs. He knew little regarding comparative medicine and his works are detestable and full of conceit and advertising. Compared with the recognized British works of the time they were far from being standard. He is to be remembered for the establishment, in 1794, of the "Equestrian Receptacle," an imposing brick building situated in London, which served as a veterinary hospital. It is interesting to learn that he announced two years after it was established that up to that time he had not lost a case. Though we intend to discuss only purely American facts, it is deemed necessary to enlarge on Taplin in order to give a true idea of the type of literature that was first thrown, figuratively, upon the unsuspecting American public. A copy of Taplin's edition is to be found in the Surgeon General's library.

In 1816, J. Carver[13] published in Long Island a book under the name, "Notice of His Horse Infirmary, and Comparative Anatomy of Man and Horse." We have not seen this work although a copy is to be found in the Surgeon General's library. The same author was again heard from two years later.

What is believed to be the first strictly veterinary record made in America is to be found in the registry of the clerk (D. Caldwell) of the Eastern District of Philadelphia. It is recorded that on June 23, 1818, "James Carver hath deposited in this office the title of a book the whereof he claims as author in the words following: 'The Farriers Magazine or Archives of Veterinary

Science, Containing the Anatomy, Physiology, and Pathology of the Horse and other Domestic Quadrupeds, and compiled from the lectures, and practices of Veterinary Colleges of London, France, Germany, Russia and British India—James Carver, Veterinary Surgeon, Master of Equitation and Corresponding Member of the London Veterinary Medical Society and the College of India.'" This is a book of a little over 100 pages containing, in addition to what the title tells us, an appeal to the people of Pennsylvania, to the public at large, a dedication and an advertisement. In the appendix Carver announces that he is going to publish a series of books, on stable duty, on food, on labor, on epidemic diseases and on many other subjects. It does not appear, however, that these books ever came to light.

Nothing more seems to have been written until 1831. From this point on, veterinary literature appeared regularly, showing that there was a demand and indicated that interest was becoming more pronounced on the subject.

Thus we note that in 1831, R. H. Budd, M. R. C. V. S.,[14] published a little treatise called "Practical Treatise on Diseases of the Foot." This little work reached a second edition in New York and had a good sale in England. It was well worth publication, remaining up to date years after Budd had passed.

J. Haslam,[15] in 1832, published a book in Baltimore entitled, "A Few Brief Observations on the Foot of the Horse and Shoeing."

In 1834, there appeared in New York the first American edition of "The Horse-Keepers' Guide," by J. Mills,[16] and in the same year was issued "The Gentleman's Pocket Farrier," by F. Tuffnell,[17] in Boston. All of these works were printed for the information of the layman and for the most part were copied from other works.

In 1839, another American edition of a British work appeared. This was authored by J. Hinds[18] and was published in Philadelphia by T. M. Smith, who added some notes applicable to American conditions of the time. This book was titled, "The Veterinary Surgeon; or Farriery taught on a new and easy plan, being a treatise on all the diseases and accidents to which the horse is liable, etc., with additions and improvements by T. M. Smith." A copy of this work is in possession of the Veterinary Division of the University of California, and one will be found in the library of the Surgeon General, Washington, D. C. Who Smith was or what qualifications he had that prompted him to elaborate upon Hinds' production we do not know. "John Hinds," we are told by Major General Sir Frederick Smith, was not a veterinary surgeon but was really Babcock, the London publisher,

who printed some veterinary works for White. He evidently did a little plagiarizing, later issuing the original of this book under the pseudonym of "John Hinds."

The veterinary literature that appeared during the period under discussion may be divided into four classes: (1) strictly veterinary books copied from English works without due acknowledgment; (2) strictly veterinary books published in England which appeared later in this country as new editions or published with additional notes by Americans; (3) productions of an agricultural nature, such as the rearing of live stock, etc., with chapters on animal diseases and their treatment. (In passing we may state that this class was undoubtedly the most prolific, and incidentally the least worthwhile, for the author usually exceeded his limitations when he attempted to inject veterinary subjects into an otherwise good book, with the result that he had to draw upon other works whose value he was not competent to judge adequately.) (4) the veterinary columns and articles published in agricultural journals and periodicals. These latter were of a type similar to the third class mentioned above.

Beyond a doubt the best productions of the period were the several American editions of Youatt's works by various individuals. William Youatt, of London, who had attended, though he had not graduated from the London Veterinary College, was undoubtedly one of the outstanding practitioners of the first half of the nineteenth century. He was the author of several books, all of which reflected the most advanced practices of his time ably written by one of England's most practical veterinarians.

The re-publication of his works in this country was a blessing to the American public and served to dispel many of the brutal and barbarous practices then in vogue. The first of his books to be published was in 1843, by J. S. Skinner. Whatever criticisms may be directed against Skinner he must be accorded the full credit due him for this edition of Youatt. Beyond being the publisher of an agricultural journal and having been Assistant United States Postmaster, any other qualifications he may have had as a veterinary writer are unknown, but it must be admitted that he had the ability to separate the wheat from the chaff as evidenced by his choice. This book was honored by a wide circulation and served as a standard authority in this country for many years.

Later appeared Youatt and Martin, on cattle, which went into 10,000 copies and later still, Youatt, on the horse, which reached 23,000 copies. A complete review of all veterinary literature has not been attempted here for the good reason that such a research

would require many years to finish. Though many others besides those mentioned are known, yet the list of these writers is far from being fully comprehensive, so for the present we are obliged to limit our list of veterinary authors to those names already mentioned. There is, however, one other work we desire to include herein and that is the book by R. L. Allen,[19] titled, "Domestic Animals; History and Description of the Horse, Mule, Cattle, Sheep, Swine, Poultry and Farm Dogs—With Directions for Their Management, Breeding, Crossing, Rearing, Feeding, and Preparation for a profitable Market, also Their Diseases and Remedies, together with Full Directions for the Management of the Dairy." This small book, with the large title, consists of 227 pages and was published in 1847 in New York and is a good example of the type of book referred to as being primarily of an agricultural nature with the addition of veterinary sections, under class 3 above.

Allen was a well-known agricultural writer, being the author of a "Compend of American Agriculture" and other works. The veterinary parts of his book are not original but are copied from other works, in most instances without proper acknowledgment, and serve to furnish us with a view of the prevalent ideas of that age, and for that reason it is gone into more in detail than other works. It is not to be supposed that this book represents the best practices of veterinary medicine of the time as reflected by Youatt, and others, but rather the ordinary practices of the period. The book is not to be judged by the portions devoted to veterinary science for apart from that it is a fine work. We will not undertake to review anything but that part which deals with disease and its cure.

In the preface Allen tells us that "the subject of animal disease is complicated and little understood; and to properly comprehend, requires years of close intelligent study, under every advantage for obtaining the necessary information." He tells us it is "absurd" that a collection of formulas given by "unskilled" hands can be of but little value. Nevertheless he gives us "a few simple remedies for the most common and well-known ailments" and suggests that the more complex be left to nature or to the "professional farrier."

The diseases of calves are discussed under "Management of Calves." A subheading, "Diseases and Remedies," taking up almost an entire page, tells us all there is to be known on this important subject. Three ailments are discussed, namely, disordered bowels, scours and diarrhea, and "costiveness" (constipation).

For the first is recommended the following: Mix 2 dr. rhubarb, 2 oz. castor oil and ½ dr. ginger with a little warm milk or gruel. One of his prescriptions for scours is ½ pint cider, with an equal quantity of blood drawn from the calf's neck. He also recommends a little rennet in the food. "Costiveness" (constipation), we are told, is removed by giving pork broth.

Ten pages are devoted to "Diseases in Cattle." In a footnote the author tells us that besides his own experience he has drawn on the *New England Farmer*, the *Albany Cultivator*, the *American Agriculturist* and other reliable American and English works for some of the remedies mentioned. From this we may deduct that he had some actual experience of his own but depended mostly on the three agricultural journals mentioned, besides other works.

His first disease quoted is "Hoven, or Swelling of the Paunch." His cause and symptoms clearly describe tympanites or bloat. We are giving his entire list of drugs. He recommends some one of the following remedies:

A pint of gin poured down the throat.

From one to two pints of lamp or some other oil.

Strong brine.

New milk with one-fifth its bulk of tar, mixed.

An egg-shell full of tar forced down the throat and followed by a second dose, if the first fails.

A tablespoonful of volatile spirits of ammonia, diluted with water.

A wineglassful of powder (gun), mixed with cold lard and forced into the stomach.

A teaspoonful of unslaked lime dissolved in a pint of warm water, shaken and given immediately.

A pint of tolerably strong lye. In addition, he recommends, should failure attend, the use of a probang.

He certainly cannot be called a one drug man. His article on choking will interest us. Among other methods he tells us that a soft root in the esophagus may be crushed, so as to allow it to be swallowed, by holding a smooth block against it and striking it on the other side with a mallet.

Horn ail or hollow horn, he tells us, is not infrequently hollow stomach due to hard usage, stinted fare and exposure to cold. The treatment—bleed and physic, shelter and feed. Take one-half pint of good vinegar, two tablespoonfuls of salt, one teaspoonful of pepper, mix and pour into each ear, holding the head to one side for two minutes. Bore with a large gimlet on the under side of the horn, three or four inches from the head, and, if hollow,

bore nearer the head, and let out all the matter, and syringe two or three times a day with salt water, or soap suds, or salt and vinegar. Spirits of turpentine, rubbed in around the base of the horns, he tells us, will arrest the trouble in its early stages.

He further suggests pouring a teakettle of boiling water on the horns, holding them so as to prevent injuring other parts: or, pouring a spoonful of boiling hot brimstone into the cavity between the horns. Just where he means is not quite clear, but that probably made little difference in the result. He tells us soot and pepper given internally are good. Apparently, before this book left the press, some one inserted a sheet opposite the article on hollow horn. This sheet is printed on one side only and is taken from the *American Agriculturist* and qualifies Allen's statement giving sensible causes for horn ail, which is described as general weakness, and rational treatment is suggested.

Jaundice in cows, Allen tells us, is due to gallstones and cured by bloodletting and purgatives. Mad itch, he writes, is a disease of the western states. (In 1847 the western states meant the Middle West.) The victims are attended by terrible itch around the nose and base of the horns. They lick their sides and backs and jerk and hiccough until they fill themselves with wind. They froth at the mouth and die raving mad in 24 hours. Later in the century, we read some very good papers on this subject by prominent veterinarians. This is the first time we hear of it.

In order, Allen takes up bloody murrain, or red water (for which he draws from Youatt) and tells us he has treated it; hoof ail; loss of cud; scours and diarrhea; warbles; wounds; milk or puerperal fever, which he treated by bleeding; caked bag; garget; sore teats; warts; sore necks on working oxen; bites of poisonous snakes; stings of bees, hornets, etc.; forming tumor; lice and vermin, and the trembles.

Trembles, we are enlightened, causes " a most fatal disease" in the human called milk sickness. The trembles existed principally in the Wabash River region and Dr. Drake thought it due to eating poison oak. The principal symptom manifested was excessive trembling and entire prostration. Allen mentions other epizoötics as being black tongue, black foot or foot rot, etc., but he tells us nothing of them.

The diseases of sheep take another ten pages. We are advised that diseases in sheep in this country are rare, and under proper care, shelter, feed, etc., sheep will "seldom if ever get diseased on American soil." He takes up a formidable list of ailments, however, and seems to rely on bleeding and Epsom salt as a sheet-anchor treatment. He describes diarrhea or scours and dysen-

tery. For the latter he recommends bleeding. For hoven he refers us to hoven in cattle. Braxy is a serious disease causing severe losses. The treatment consists of bleeding, which sometimes is difficult on account of the stagnation of the blood. In this case immerse the patient in a tub of hot water to make the blood flow more rapidly. "Stretches" is an ailment when sheep stretch out their noses on the ground, and around to their sides, as if in severe pain. This may be caused, he says, by an involution of one part of the intestine in another. When due to this cause, jerking the sheep by the hind leg will restore health. Inflammation of the lungs is treated by bleeding.

Rot (flukes), he tells us, is practically unknown in this country but foot rot is very common. He has a long article on the latter subject, which is very reasonable. He suggests, however, among other preparations, the use of urine as a wash for the feet. In addition he treats costiveness, poison, corrosion of the flesh by flies and maggots, flies on sheep, protection from gadfly, swollen mouth, foul noses, scab, ticks and lice, pelt-rot (falling out of wool), staggers or sturdy (hydatid), and water in the head, abortion, the uterus (prolapsé), garget, and large cuts. It is apparent that Allen did not feel at ease when treating sheep. About one-half of his articles are complete reprints from *The Genesee Farmer, New England Farmer, Albany Cultivator* and *Cultivator.* He also refers to H. D. Grove and to Blacklock.

He completes his section on sheep diseases by an article from Blacklock on bleeding. It is quite complete, giving a detailed modus operandi. Of interest is the statement that for recovery bleeding must be rapid and from a large orifice. "Little impression can be made on an acute disease by slow bleeding." The jugular vein is the best choice and the animal must be bled until it falls or is about to fall. When this is accomplished a pin should be run through the edges of the cut vein, and a lock of wool twisted around it.

The section on the diseases of the horse takes up some 27 pages, giving a most formidable list of ailments. We note that colts are castrated standing. "If large and fractious he must be cast," but apparently the standing method was the accepted one. The cord is either scraped or twisted; clamps are not mentioned.

He draws heavily, usually acknowledged, on Youatt for his section devoted to horses. Consequently, in this portion of his book, he expresses, more or less, the advanced thought of his time, more so than with other animals. Glanders and farcy are known to be related, although it is still thought that glanders may be

cured in its first stages. Lampas is understood, although in aggravated cases the bars of the mouth may be lanced for immediate relief.

All those affections which the English classified as pneumonia, or inflammation of the lungs, chronic cough, thick and broken wind, consumption, etc., Allen classifies as heaves. In pneumonia he recommends free bleeding. Catarrh, or horse distemper, is recognized as infectious.

He classifies two kinds of colic, spasmodic and flatulent. For the former, turpentine, opium and bleeding are recommended and for the latter, turpentine, opium, chlorid of lime and the trocar.

Inflammation of the bowels, he states, is of two varieties. The first is an inflammation of the external layers of the intestines and the second variety an inflammation of the internal coats. Bleeding, of course, is resorted to in the first type, removing six to ten quarts of blood, "in fact, as much as the horse can bear." This should be repeated if no relief is evident, removing four to five quarts more. In the second variety, bleeding is rarely necessary. Further treatment in both cases is more or less rational.

Two pages are devoted to physicking. This phase constitutes almost a major operation, including preparation for, the actual administration and after care, requiring from three to five days. It is to be repeated in a week if the animal is not too weak from the first administration.

This is followed by a long list of foot ailments, such as ringbone, curb, bone spavin, grease, etc. This part of the book is particularly good, but it is to be remembered that the greatest advance made by the early school under Gibson, Osmer and others of the eighteenth century was in lameness.

Founder treatment is taken from Youatt, and includes the taking of four quarts of blood from each toe.

Regarding epizoötics in horses, Allen tells us they "sometimes occur." One occurred, he states, in 1846 in and around New York, termed "a malarious congestive fever, staggers or apoplexy," which caused the death of many valuable animals. The most effective remedy used at this time was taking a quart of blood from the head, swathing the head with cloths saturated with cold water and giving two drams of calomel daily. The only postmortem lesion to be found was a mass of congested blood in the brain.

The diseases of swine are confined to pulmonary affections, colds, coughs and measles (of which he tells us nothing besides the names), costiveness (constipation), itch and kidney worms.

Bleeding is directed in pneumonia and kidney worms are considered incurable. To blind staggers he allots a separate heading. Bleeding is resorted to. He then tells us how to bleed and adds that the diseases of hogs have received little attention and the treatments are more or less haphazard.

Of the treatment of dogs he tells us nothing.

To hens he devotes one page and deals with gapes or pip, roup, catarrh or swelled head, flux, costiveness, and vermin. Gapes or pip, we are told, is due to drinking dirty or unwholesome water. The treatment is interesting. "Remove the white blister on the tip of the tongue, and wash with sharp vinegar, diluted with warm water; or compel the bird to swallow a large lump of fresh butter, mixed with Scotch snuff. It has been cured by opening the mouth and forcing a pigeon feather, with a tuft of feathers left on the end (the others having been stripped off), down the windpipe and gently turning it as withdrawn, to be repeated the following day if necessary." This, we are told, detaches large numbers of red worms in the larynx that interfere with swallowing and breathing. He knows roup is infectious and advises separating sick ones at once.

When finishing his chapter on diseases of cattle, he tells us what is probably the prevailing idea of his time among intelligent people regarding the farrier. He suggests that if "intelligent farriers" are to be had they may be called on with advantage. This definitely tells us that all farriers, in his estimation, and he is probably correct, were not intelligent. He acknowledges his distrust of the quackery of those passing under the title. He says they are mostly empirics with little science and intelligence in this class the world over. Here he was reaching a little too far. He advises the owner to see to it that, if he employs a farrier of whom he is doubtful, no medicines or practices that are severe or hazardous be used, particularly the indiscriminate use of poisons, boiling oils, turpentine, tar and the hot iron.

The review of this book has been long, and perhaps irksome to the reader, but it has been given for the purpose of showing the practice of the day. Not the best, as taught by Youatt and others, nor yet the worst, as given by some of the poorer works of that period, but that which the average farmer would most likely get and follow.

After reading this we naturally wonder what type of men constituted the veterinary profession at the end of the period under discussion, viz., 1850. They appear to be of three distinct types: The most numerous by far was the common or "hereditary"

farrier who was self-styled and either inherited his learning, or rather his lack of it, from his father, or else picked it up in the stable. The second class was originally the same as the first but was earnest and studious and made the best of circumstances to qualify himself. This man, though denied the advantages of an education that was not obtainable in this country, learned surprisingly from a few textbooks and by close observation. He took his work seriously and was a real credit. He existed in too few numbers, but was really the backbone of the profession. The third type of men, probably not more than ten in number at this time, were the graduates of foreign colleges or physicians practicing veterinary medicine, of whom there were several. These men stood apart by themselves, but being only a handful they naturally settled in the larger cities, such as Boston, New York, Baltimore and Philadelphia, and consequently their influence was limited to those places.

Probably 95 per cent of the men who turned their attention to the treatment of animals were the common farrier, first spoken of, hence his influence cannot be discounted. In the first part of the century he was probably the blacksmith, horse-trainer or stable groom. As the years passed by, the live stock interests became more valuable, both in numbers and as individuals. This encouraged the migration to this country of a few men trained, or partially trained, in animal pathology. These latter, by showing their superior skill over the stable groom, and his ilk, won for themselves remunerative patronage. Their economic success encouraged empiricism and charlatanism, those gaunt spectres who spread throughout the entire country and have persisted in stalking the profession until recent years and whose hollow moans can even yet be heard on occasions.

Many of the depraved, drunken and disreputable horse traders, anvil artists and livery stable parasites, whose principal qualifications were greed and dishonesty, decided to call themselves farriers and veterinary surgeons, often assuming that title overnight. Many of them inherited their desire and so-called attainments from their fathers, men like themselves, and felt thus well qualified to prey upon the public. With the increase in numbers and value of live stock, their numbers increased also, not in proper proportion, however, but in leaps and bounds. The public, unaccustomed to scientific veterinary practices, fell unwittingly into the hands of these unscrupulous mountebanks whose chief stock in trade was a glib tongue and their asserted ability to cure "hollow horn," "wolf in the tail," "chest founder," "loss of cud," "moon eyes," "hooks," and a host of other ills, real and

imaginary. From these men the general public, ignorant as to the qualifications that a scientific doctor of animals should possess, began to think of them as the true representatives of the veterinary profession, that veterinarians were anything but gentlemen, and certainly were not fit to travel in ordinary social circles.

Some of the more intelligent stock-owners were very much dissatisfied with these frauds and believing that the ailments of animals should be ameliorated by less brutal and more rational methods of treatment began to refer to the popular veterinary books of the time, those of Taplin, Clater, Youatt and others. They slowly became more or less conversant with their contents and, by application and observation, attained some skill, which in turn they gave to the neighborhood. Here we see the rise of the second class mentioned above.

This phase of development is best expressed by one of these men, Albert Burroughs, of Kirby, Vermont. He wrote a letter to Dr. G. H. Dadd, of the *American Veterinary Journal*, early in 1852. Dadd printed it in his journal, and, in our opinion, it is well worth reproducing.

> I commenced the treatment of disease in horses and cattle from sheer necessity and without much preparatory instruction. When a boy, I had under my care, during the winter months, from 40 to 60 head of cattle, a hundred sheep or more, and from 6 to 8 horses and colts; these, of course, were sometimes out of condition, and subjects of the prevailing maladies which were more or less severe according to circumstances. Having no farrier in the vicinity, my mind was naturally employed devising ways and means for the welfare of my charge—the prevention of disease and its cure. Having no means at hand for the treatment of disease, except the simple agents procured from nature's garden, these I found efficient; experience gave me confidence, my practice was successful. My success gave me notoriety among the neighbors, and those having sick animals were sure to avail themselves of my unpretending services. In this manner the professional honors have been thrust upon me. I have, however, endeavored to qualify myself for the task by studying the best works to be found on the veterinary art; but as the treatment of sick animals is but a small portion of my business, I can not give it that attention which the importance of the subject demands. Its importance is exemplified in the following cases:
>
> Last summer, having occasion to absent myself from home for a short time, I found, on my return, that a valuable cow had suffered some from the hands of a "traditionary" farrier who bored the cow's horns for horn ail, the stomach was scoured out with a strong drastic purgative, a garget rowel was inserted in the breast; the tail and ears were cropped "to start the blood," etc., etc. In this manner the poor animal was treated, until death ended the scene.
>
> I was called to see a mare, a short time ago, the subject of a severe and marked case of colic. A number of persons being present, they, according to custom, offered their opinion. One man suggested that "the mare had the bellyache," and ought, therefore, to

> have a good nipper of gin and molasses. Another individual, perhaps somewhat in favor of the Maine liquor law, recommended one ounce of laudanum and half a gill of spirits of turpentine. "She has got the bots," says the grocer, "give her a pound of green tea." "No," said another, "it's inflammation of the guts; give her a quarter of a pound of aloes." And so they continued to guess and prescribe, to the end of the chapter.

He continues by giving his treatment. The animal being in an exposed condition, rolling in the snow, he clothed the animal with blankets, rubbed the belly and legs, applied four hot bricks to the belly, gave two quarts of mint tea and administered an injection of warm water, and followed this up with a warm infusion of linseed oil. The animal recovered, or he would not have written about the case, obviously.

Further than what is reflected in this letter, we know nothing of Burroughs, except that he contributed another letter to the *American Veterinary Journal*, wherein he advocated the necessity for better education for the veterinarian and the elimination of the ignorant class dominating the profession, and also the correcting of brutal practices then in vogue by that class.

We do not know of many individuals, or personalities, connected with the veterinary art during this period, but a few names and incidents have come to us through various sources.

Earliest Graduates in America

We are told by Sir Frederick Smith that, in 1817, Bracy Clark, one of the prominent veterinarians of the period, in England, became very ill. Shortly after this his nephew, Charles Clark, came from America and went into practice with him as a partner. Later, about 1828, when Bracy Clark retired from practice, Charles took over the entire work. Knowing of Bracy Clark's qualifications—he had been a surgeon, was a graduate of the London Veterinary College, a successful veterinary practitioner, a voluminous writer, a man, also, with an excellent classical education, particularly in Latin and Greek—we hesitate to believe that he would take as a partner any one who was not qualified, or at least partially so. Smith makes it very clear that the younger Clark was not an apprentice or an assistant. From this we must assume that we had in America, prior to 1817, at least one qualified, or at least partially so, veterinarian in the person of Charles Clark.

The first European graduate, apart from Clark, whose qualifications we can only assume, to settle on our shores was John Rose. He was a Prussian graduate and settled in New York in the year 1827. Unfortunately, beyond this fact and that he commanded a large practice, we know nothing of him.

In the same year that Rose came to the United States one Saunders came to America from England. Of Saunders we know little except that he obtained a position as veterinarian with the Old Eastern Stage Company in Massachusetts, which position he held until his death. He was, however, the father of W. Saunders, who became prominent as a veterinarian some years later. The junior Saunders' greatest activities extend to later periods and will be discussed in their proper sequence.

At this very period (1827), the leading practitioner in Boston was a Mr. Harrington, a blacksmith. Harrington treated sick horses as a part of his blacksmithing business, and enjoyed a large patronage. It appears that he specialized in theory and practice, leaving the surgery to his workman, John Davis. The latter was often seen on Sundays with his firearms and bleeding stick practicing the art of venesection. Skilled in their art as they were, once in a while a case presented itself that seemed obscure to them, baffling both the physician and the surgeon. These cases usually were sent to a Dr. Brown, who had a large infirmary in Roxbury, but beyond this we know nothing of Brown.

The Micheners

About the year 1830, Isaiah Michener (born about the year 1811) began practice in Pennsylvania. Dr. Michener was a very prominent and respected practitioner in Buckingham, Bucks County, of that state, where he engaged in practice for upwards of half a century. He contributed to the first veterinary periodical of this country *(American Veterinary Journal)*, and Dr. George Dadd included in one of his books ("The Modern Horse Doctor") an article by Michener on "putrid fever," a disease he had encountered in Pennsylvania. From his article it can be seen that he had considerable education, as he expresses himself very clearly and in very fine English. In addition, it is clear that he was a rational thinker, a close student, and one anxious to learn by inquiry. In an article printed in the *Bucks County Intelligencer*, issue of November, 1845, the editor tells us, as an introduction to a letter written by Michener, through the paper, to a client of his, on the subject of putrid fever, that he (Michener) "is one of the most successful farriers of our County."

He was father of C. B. Michener, one of the first American graduates who later became secretary and president of the United States (now the American) Veterinary Medical Association, besides otherwise distinguishing himself. It has not been made clear as to whether or not he had received any preparatory veterinary education, but it is assumed not. In any event he must be

considered one of our outstanding pioneers. He was a kind and genial man, respected by his clientele and his professional brothers. He was an ardent supporter and member of the American Veterinary Association of Philadelphia and when that body established the Pennsylvania Veterinary College, to supplant the Philadelphia Veterinary College, he was a member of its faculty in the capacity of Professor of Theory and Practice of Medicine and as a clinical lecturer.

When the United States Veterinary Medical Association was organized in New York, in 1863, the selection of a name presented a delicate problem, as already there existed in Philadelphia an organization called the American Veterinary Association. Dr. Michener came to the rescue by suggesting "United States" as the name of the national body. He was also elected corresponding secretary at its first meeting. Michener remained faithful to the U.S.V.M.A. and as late as 1891, at the age of 80 years, gave a most interesting and entertaining address before that body at its annual meeting in Washington, D. C.

R. H. Budd, M.R.C.V.S., of London, landed in New York in 1831, where he combined the horseshoeing business with his practice. He left a little work called, "Practical Treatise on the Diseases of the Foot," which has already been discussed.

Chas. M. Wood

In 1835 Chas. M. Wood started practice in Boston. He was born about the year 1797 and though we have not been able to learn anything of his earlier life and education it is evident that his preliminary preparation was thorough. Nowhere do we find evidence that he attended any medical or veterinary schools but his writing and general demeanor give the impression that he was not only far above the average man of his day but they betray signs of a professional scholar.

He stood in a class by himself as a veterinarian and as such received the honor and remuneration of an extensive practice. He was an intelligent man of high principles. He served as Professor of Theory and Practice of Veterinary Medicine in the Boston Veterinary Institute and was active in the organization of the Boston Veterinary Association, serving as its first president. As an acknowledgment of his erudition he was granted the honorary degree of M. D. in 1857, by the Eclectic College of Medicine of Cincinnati, Ohio. This honor came to him on account of his connection with the Boston Veterinary Institute. He wrote many articles for the *American Veterinary Journal* and all of them are worthy of attention. One in particular, on "Veterinary

Medical Associations," written in the fifties, is one of the best articles on the subject we have ever read.

Dr. Wood was very active in the organization of the United States Veterinary Medical Association (A.V.M.A.), serving as its second treasurer (1864-1865) and its third president (1865-1866). We have not been able to ascertain the date of his death.

In 1836, certain disorders by students against the faculty took place in the London Veterinary College. The ringleader of the students was a William Miles, an American, of New York. This is a significant fact. It shows us that sufficient interest was being shown in this country in veterinary matters to cause at least one American to go to Europe, as early as that year, to qualify himself as a trained veterinarian. Miles did not return to this country but remained in England. He subsequently wrote some books. "The Horse's Foot and How to Keep It Sound," from the third London edition, was published in New York, in 1847. It was the perusal of Miles' "Practical Farriery," published in 1886, and which contains a brief early history of the veterinary art, that interested the present writer in veterinary history.

In 1837, C. C. Grice, a graduate of London in 1826, came to New York and later R. H. Curtis, A. Lockhart and C. Pilgrim, all graduates of the London school, settled in that city. These men, with Budd, may be called the pioneers of veterinary medicine in New York State.

In the last decade of this period, that is, between 1840 and 1850, we meet with quite a number of names of men who were in practice at that time. Among them we find that of W. H. Lillyman, who was graduated from the London Veterinary College, in 1840. He came to Boston shortly afterwards and enjoyed a fine reputation. Dr. Dadd, a competitor, conceded that he was a clever man of much experience and we take this to mean a whole lot, as Dadd wasted little ink lauding anyone save himself.

In Lowell, Massachusetts, was Robert Wood, a man of excellent reputation. In 1847, George H. Dadd, M. D., turned to the practice of veterinary medicine in Boston, and in Utica, New York, was to be found A. S. Copeman, eminently qualified as practitioner and teacher. He was supposed to be a graduate of London, although there seems to be some doubt about it. The activities of these men, as in the case of Grice, Curtis, Lockhart and Pilgrim, were exerted at later periods, and will be discussed in their proper place.

In Philadelphia, T. J. Corbyn was by far the leading man in the forties. It was under Corbyn that Jennings, of whom we are

to hear more later, studied about the year 1840. Also in that city were W. W. Fraley and John Scott.

It will be seen that at the close of the period several names of men who were to distinguish themselves appeared. The middle of the half-century then finds us with a nucleus of distinguished men prepared to advance both the art and the profession. We are now ready, using the term that Sir Frederick Smith employed, for the analogous period in England, around the year 1790, to enter upon the transition period.

REFERENCES

[1]Liautard, A.: The history of veterinary medicine. Amer. Vet. Rev., 1 (1877), 1, pp. 1-19.
[2]Huidekoper, R. S.: Amer. Vet. Rev., vii (1884).
[3]Huidekoper, R. S.: Jour. Comp. Med. & Surg., x (1889), pp. 332-351.
[4]Houck, U. G.: The Bureau of Animal Industry of the United States Department of Agriculture. (U. G. Houck, Washington, D. C., 1924.)
[5]Eakins, H. S.: Vet. Bul., xv (1925), 5, 6, 7 and 8; xvi (1926).
[6]Sayre, F. B.: Observations on the disease commonly called yellow water in horses. N. Y. Med. Repository, iii (1800), 1st series, pp. 312-344.
[7]Barton: Some account of the disease called the hollow horn. Med. & Phy. Jour., i (1805), pp. 77-79.
[8]Stevenson, J.: Case of successful treatment of yellow water. Philadelphia Med. Museum, iv (1808), pp. 35-42.
[9]Smith, J.: Account of a disease among neat cattle apparently caused by browsing the juicy sprouts of oak shrubs, in the woods of Suffolk County, New York. N. Y. Med. Repository, i (1809), 3rd series, p. 38.
[10]Jewett, Paul: The New England Farrier. (A. Stoddard, Hudson, N. Y., 1806.)
[11]Livingstone, R. R.: An Essay on Sheep. (London, 1809.)
[12]Taplin, Wm.: The Gentlemen's Stable Directory, etc. (Philadelphia, 1812.)
[13]Carver, J.: Notice of His Horse Infirmary, and Comparative Anatomy of Man and Horse. (Long Island, N. Y., 1816.)
[14]Budd, R. H.: Practical Treatise on Diseases of the Foot. (R. H. Budd, New York, 1831.)
[15]Haslam, J.: A Few Brief Observations on the Foot of the Horse and Shoeing. (J. Haslam, Baltimore, 1832.)
[16]Mills, J.: The Horse-Keepers' Guide. (New York, 1834.)
[17]Tuffnell, F.: The Gentleman's Pocket Farrier. (Boston, 1834.)
[18]Hinds, J.: The Veterinary Surgeon; or Farriery, etc. (T. M. Smith, Philadelphia, 1839.)
[19]Allen, R. L.: Domestic Animals; History and Description of the Horse, Mule, Cattle, Sheep, Swine, Poultry and Farm Dogs, etc. 227 pp. (New York, 1847.)

Iowa Veterinarians Show Horses

Well-known Iowa veterinarians were among those acclaimed at the first annual American Legion Horse Show, held at Marion, Iowa, June 10, 1934. Dr. H. N. Strader, of Marion, rode his own horse in one entry, and the saddle horse owned by Dr. F. M. Wilson, of Mechanicsville, was the winner of a prize. Dr. C. A. Bradley, of Marion, entered "Skipper," a pony, which was also a prize-winner. Dr. V. G. Bromwell of Center Point, served as official veterinarian of the Show. Society turned out well, and the affair created much interest and was a decided success.

J. B. B.

FURTHER STUDIES IN THE CONTROL AND HOSPITALIZATION OF CANINE DISTEMPER*

By M. L. MORRIS, *Stelton, N. J.*

The control and hospitalization of distemper always has been one of the most difficult problems associated with canine practice. With the introduction of vaccine prepared according to the findings of Laidlaw and Dunkin,[1-5] prophylaxis against this disease was greatly stimulated. By the application of laboratory methods and the use of nucleotide[6-8] in addition to the employment of products as described by Laidlaw and Dunkin, we have been able to decrease the mortality rate in dogs afflicted with this disease.

Before attempting an individual analysis of case records, it may be well to consider briefly the morphology of the various blood-cells to be discussed in the blood-pictures which follow. The genesis of these cells is well explained by Schilling,[9] Piney,[10] Kolmer and Boerner,[11] and others. For this study they may be classified into the following groups:

I. Erythrocytes, or the red cell system.

II. Leucocytes, or the white cell system.

1. Neutrophiles: Composing this system are the immature and mature neutrophiles. The immature neutrophiles are made up of the myelocytes, juveniles and stabs.

2. The lymphocytes are composed of two varieties—large and small.

3. The monocytes, of which the large mononuclear cell is the representative.

The basophile does not occur commonly in the blood of the dog, and I am not familiar with its importance.

The eosinophile, however, is a more evident cell, appearing in variable numbers, rarely exceeding 10 per cent in a given count, although counts of 20 to 30 per cent have been seen. We do not feel that this cell serves as a reliable index to parasitism, because it is often entirely absent in heavily infested dogs.

The myelocytes are not a numerous cell and when appearing in the blood of the dog are evidence of a very acute infection. The juvenile, a more mature cell than the myelocyte, is present quite frequently but rarely occurs in large numbers. The stab cell is a very prominent cell in the blood of the dog and may reach 70 to 80 per cent of the total in hyperacute infections. The segment, or mature polymorphonuclear, does not occur in as large numbers

*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

as might be supposed and likewise may disappear to the low point of 5 or 6 per cent in dogs seriously ill.

The lymphocytes appear in the blood of the dog in large numbers. In young, healthy, rapidly growing puppies, these cells may constitute 75 to 80 per cent of the total. They likewise are an important cellular constituent from the standpoint of prognosis in canine infection. The mononuclears rarely appear in large numbers and their presence is considered by some hematologists as a favorable omen. We will now consider the relationship of these cells in the average normal blood-picture of a six-month-old dog.

The average red blood cell count of a good healthy growing puppy at six months of age will approximate 6,000,000 to 7,000,000. It is very common, however, to see red cell counts of from 3,500,000 to 5,000,000 in undernourished, or parasites-infested dogs.

The hemoglobin will more commonly be seen below the range of 94 to 105 per cent than above. In dogs heavily infested with hookworm this factor will often drop to 35 per cent. Likewise dogs fed on narrow rations, especially those deficient in meat and minerals, also fail to meet the normal hemoglobin standard. Dogs deficient in erythrocytes and hemoglobin are always more difficult patients than good normal red-blooded pups.

The white blood cell count also is a somewhat variable factor but usually falls within the 10,000 to 14,000 range. Dogs transported to the hospitals in automobiles, following a heavy meal, may show excessively high white counts and in such instances they should be rechecked the following day.

It is uncommon to see more than a single basophile in a given count, but the eosinophile count may vary from 0 to 8 or 10 per cent, with an average of from 2 to 5 per cent. It seems peculiar that this cell does not occur more regularly, since dogs are so commonly infested with parasites. It is possible, in certain young, rapidly growing pups, to find normally 1 or 2 per cent of myelocytes, but their presence in any numbers would suggest an acute infection.

The juvenile, another immature cell, may be totally absent, but the appearance of the cell to the extent of 2 or 3 per cent in a given blood-picture would be considered normal. The stab cell more nearly approaches the mature segment, and constitutes 20 to 35 per cent of the normal differential count. It is not infrequent to find 12 or 14 per cent of these cells, but if they occur in increasing numbers, it is usually indicative of an infection.

The mature segment usually appears to the extent of from 40 to 60 per cent. This figure, however, may not run so high in young dogs whose immature and lymphocyte counts are increased. The lymphocyte count is quite variable, even in puppies of the same litter, but an average count of 36 to 50 per cent is considered approximately normal. The mononuclear is often characterized by its total absence, but in a given count from 2 to 4 per cent would not be considered as abnormal.

Certain characteristic changes seem to occur as the age of the dog increases. Most notable is the decrease in the total white blood cells, lymphocytes, myelocytes, juveniles and stabs, with a gradual increase in the segment count. These changes seem to continue until maturity.

Considering the blood-picture as a whole, certain deviations from the normal may be expected, depending upon the age, breed, sex, method of handling, individual disposition, composition of the food, exercise and climatic conditions. There will also be noted a slight variation in blood-pictures reported by different technicians. For the most consistent practice, counts performed by a single technician are desirable.

With this clearly in mind, it is evident that the nuclear index, as described by Gerard and Boerner,[12] can be subject to quite a wide variation and still be within the normal range. This index is arrived at by dividing the total segment count by the total immature count. We note, therefore, that the nuclear index for a dog of this age would be 1.8 to 3.0. It must be kept in mind, however, that certain individuals will fall slightly below this figure, occasionally to 1.2 to 1.3, and still be healthy. When such counts appear in puppies presented for immunization, I usually examine them very carefully and often request a recheck five to seven days later.*

With this blood-picture clearly in mind, we will now attempt to analyze and interpret some blood-pictures on dogs as they are regularly presented at a veterinary hospital, or as they may occur in daily practice for examination, diagnosis and treatment.

Our first patient (see table I) will be a Chow, male, nine months old, well grown, in good coat, and a fine specimen. He had an entropion operation as a young puppy and was first presented to us with a badly broken claw. We were asked to examine the eye, as it was occasionally a little congested, care for the claw, and administer canine distemper vaccine. The tempera-

*All differential counts comprising this series are compiled on a filament, nonfilament basis.[13] The nuclear indices, therefore, are lower than in series where knobby forms are classified as mature neutrophiles.

TABLE I—*Early virus blood-picture—male Chow, nine months. (R. B. C., 5,550,000; hemoglobin, 71 per cent (12 gm); feces, negative).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Mar. 27	13,200	32	34	34	0	1.0	101.5	None	None	30 cc H. S.
28							101.4	Good	Good	
29	12,250	53	34	13	0	0.6	101.6	None	Good	20 cc H. S.
30							101.2	None	Good	20 cc Nu.
31							101.4	Good	Good	24 cc Nu.
Apr. 1	12,800	52	29	18	1	0.5	100.4	None	None	30 cc Nu.
2							101.6	Good	Good	20 cc Nu.
3							101.2	Good	Good	15 cc Nu.
4							102.0	None	Poor	Fluids
5	14,000	38	27	34	1	0.7	101.8	Good	Fair	
6							101.0	Good	Good	15 cc H. S.
7							100.0	Loose	Good	
8							101.0	Soft	Good	20 cc Nu.-Fl.
9							100.8	Soft	Good	
10	12,100	41	21	36	1	0.5	102.0	Good	Good	
11							101.0	Soft	Good	20 cc H. S.
13	13,050	47	26	26	1	0.5	100.6	Good	Good	
18	13,600	42	28	28	2	0.6	101.0	Good	Good	
24	14,000	38	44	16	1	1.1	101.5	Good	Good	Discharged

Note: Restraint difficult; treatment limited.

H. S. = Homologous serum
Nu. = Nucleotide
Fl. = Fluids

R. B. C. = Red blood cells
Im. = Immatures
Seg. = Segments

Lym. = Lymphocytes
Mon. = Mononuclears
N. I. = Nuclear index

ture was 101.5° F., appetite and bowels were reported in perfect order, and the dog was and had been feeling very well. A sample of blood was drawn from the saphenous vein and slides were prepared for laboratory examination. The count was completed while we cared for the claw and eye. We prepared to give the vaccine but, before doing so, requested the blood report. The white count was 13,200 with 32 immatures and a nuclear index of 1.0. The red count of 5,550,000 and the hemoglobin of 71 per cent were subnormal. The laboratory report on the stool was negative for intestinal parasites.

The dog was apparently not in a normal condition, although he looked to be a splendid specimen and a picture of health. On questioning the owner, I found that she had also a Chow female at home with five beautiful pups eight weeks old. This Chow was held in isolation and the female, with her puppies, was brought in for examination the following day. Suffice it to say that a diagnosis of canine distemper was made on the female, and the puppies were treated with homologous serum and returned to the owner. The male and female were treated and entered the hospital. I am showing only the blood-picture on the male, to emphasize the importance of this procedure in this case.

I would now like to call your attention to the second and third counts and especially note the rapidly falling nuclear index and the absence of the typical temperature. Following the second blood count, this dog was placed in the distemper ward with the female which did show some rise in temperature and more definite clinical symptoms. The symptoms in the female were thought by the owner to have been due to the puppies suckling. Both dogs were treated with homologous serum and nucleotide. They made a complete recovery and the pups did not develop the disease. I cannot help but think of the situation had we administered a dose of distemper vaccine and allowed the male Chow to go home. The owner, a prominent breeder, would at once have blamed the inoculation for the break.

Again referring to table I, you will note that the nuclear index again was approaching normal when the dog was discharged. I feel that, in view of the clinical history and findings, with a rise in the total white count, a lowering of the nuclear index, and the later development of clinical symptoms, to place this as an early virus picture is indicated.

Case 2 (table II) is similar to that of the Chow, but of a little different nature. It is also an early virus picture. I will use

TABLE II—*Early virus blood-picture following canine distemper vaccine—male Schnauzer, nine months. (R. B. C., 6,100,000; hemoglobin, 87 per cent (15 gm); feces, negative).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Apr. 6	15,000	39	44	17	0	1.1	100.5	None	None	30 cc H. S.
7	15,000	36	41	23	0	1.1	100.2	None	Good	
8							100.4	Good	Good	20 cc H. S.
9							101.0	Good	Good	20 cc Nu.
10	13,800	38	35	26	1	0.9	100.8	Good	Good	20 cc Nu.
11							100.6	Good	Good	20 cc H. S.
12							101.0	Good	Good	20 cc Nu.
13	16,900	46	37	16	0	0.8	101.0	Good	Fair	20 cc Nu.
14							101.2	Good	Good	20 cc Nu.
15							100.6	Good	Good	15 cc H. S.
16							100.5	Good	Good	
17							100.6	Good	Good	
18	14,000	40	33	27	0	0.8	100.4	Good	Good	
20	11,800	36	38	25	1	1.0	100.8	Good	Good	Discharged

as an example of this type of case a Schnauzer, male, nine months old, recently purchased at private sale. The dog was well grown, in fine coat, active, with normal appetite and temperature. He had been in this owner's possession for the past three weeks and had shown absolutely no symptoms of illness. This was an ambulatory case, 30 miles from the hospital, and if a blood count had been made it would have been necessary to return to vaccinate the dog, which, in view of the history, seemed very unnecessary. Therefore, a 5-cc dose of distemper vaccine was administered on March 31.

On April 6, the owner called by phone and complained that the dog was not well. He had refused his food and would not stir from his house. Thinking that possibly an abscess may have formed at the site of injection, I requested that the animal be brought to the hospital for examination. On arrival, we found that this was not the case. The temperature was elevated to 103.5° F., the eyes were a little congested and watery, and he had vomited his previous meal. Again referring to the table, we find a white count of 15,000 and a nuclear index of 1.1. Since the dog had ridden a considerable distance in a car, I advised that he be held in isolation until the next day, when a second count could be made. It is interesting to note the consistency of the two counts, the total white counts being the same and also the nuclear indices. The immature count is 3 per cent lower, also the segments, but the lymphocytes are increased 6 per cent. With these minor changes we still have a nuclear index of 1.1.

Now note what occurred three days later. The white count is going down and also the nuclear index. The diagnosis was canine distemper and the dog was placed immediately in the ward. Copious doses of serum were administered, as indicated in table II. Nucleotide also was given, although it may not have been necessary in such a case. The Schnauzer made a prompt recovery in two weeks and reports in June and July were favorable.

This case, I feel, explains some of the breaks we get with canine distemper vaccine. The product is really not at fault, nor is the technic of the veterinarian in administering the vaccine. The dog has apparently been exposed and is about ready to come down when a dose of vaccine is given. It appears to me that the vaccine only hastens the activity of the virus and these dogs often become very sick and the result is a high mortality. I have found that the blood-picture, with carefully selected histories and clinical observation, in many instances prevents the embarrassment of immunizing an already infected dog.

TABLE III—*Early clinical distemper—male Airedale, six months. (R. B. C., 4,320,000; hemoglobin, 71 per cent (12 gm); feces, hookworm, ascarids).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Feb. 14	14,750	15	22	63	0	1.5	101.4	None	None	
15							101.4	Good	Good	20 cc H. S.
16	10,650	42	21	34	0	0.5	102.0	Good	Good	
17							101.4	Good	Good	20 cc Nu.
18	12,800	23	24	53	0	1.0	100.8	Good	Good	30 cc Nu.
19							102.0	Good	Good	30 cc Nu.
20	10,600	30	30	40	0	1.0	101.4	Good	Good	30 cc Nu.
21							101.2	Loose	Good	30 cc Nu.-Fl.
22							101.2	Loose	Good	30 cc Nu.-Fl.
23	10,000	31	27	43	0	0.8	101.8	Loose	Good	30 cc Nu.-Fl.
24							101.6	Good	Good	
25	13,400	29	28	41	1	0.9	101.8	Good	Good	Discharged
Apr. 18	9,100	20	33	47	0	1.6	101.5	Good	Good	Parasites

Passing from the problem of an immunization break to that of the dog with which all small-animal practitioners are familiar, we will consider the case of an Airedale (table III), male, six months of age. He was not feeling well, lacked his usual pep, seemed to have chills, eyes were a little sore and it was necessary to clean them two or three times a day, but they refilled. The dog possibly had worms. The temperature was 101.4° F., with a stool positive to hookworms and ascarids. The clinical symptoms were not sufficiently in evidence to justify me in immediately placing this dog in a distemper ward. Examination of the blood-picture did not give the assistance that it might. Although the total white count was up to 14,750, the immature count was but 15 and the lymphocyte count was high.

The dog was placed in isolation and a second count taken in two days, which showed a marked change with a lowered white count and a nuclear index of 0.5. Clinical symptoms were more in evidence, with a slight nasal discharge, and the dog was moved at once to the distemper ward for treatment. Homologous serum, followed by nucleotide, as indicated in table III, was the only treatment given and the dog was discharged at the owner's request, with a guarded prognosis. He was not returned for parasite treatment until April 18, at which time the last count was made. A nuclear index of 1.6, although a little low, was considered normal, since the dog had consistently run a high lymphocyte count.

The owner reported that the dog had a slight cough and did not feel well for eight to ten days after he went home, but following that seemed to regain his health rapidly. This dog at no time had a temperature curve typical of the disease and, except for the eye and nose symptoms, little clinical evidence was available. It occurred to me that possibly such an animal did not have distemper. With this thought in mind, definite contacts were allowed on three occasions, with no visible effect. These are the cases that have always been perplexing and the ones that can easily contaminate a non-contagious section.

We will now look at a little different type of clinical case from the angle of diagnosis. This was a black mongrel (table IV), male, 18 months old. He made his home on the same estate with other dogs that I happened to know were sick with distemper. This patient was presented at the hospital on the evening of April 30, with the history of having had a cold for ten days or two weeks. The temperature was 103° F., some nasal and eye discharge was evident, and forced respiration could be noted easily.

TABLE IV—*Clinical distemper with secondary infections—male mongrel, 18 months. (R. B. C., 4,990,000; hemoglobin, 86 per cent (14.8 gm); feces, negative).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Apr. 30	10,750	44	22	27	0	0.5	103.0	None	None	20 cc H. S.
May 1							102.0	Soft	Good	20 cc Nu.-Fl.
2							100.8	Good	Good	20 cc Nu.-Fl.
3							100.6	None	Good	20 cc H. S.
4	9,100	38	32	21	0	0.8	100.0	Good	Good	20 cc Nu.-Fl.
5							100.6	Good	Good	20 cc Nu.-Fl.
6							101.0	Good	Good	20 cc Nu.-Fl.
7							101.0	Good	Good	20 cc Nu.-Fl.
8	12,200	48	29	21	1	0.6	101.0	Good	Good	20 cc Nu.-Fl.
9							100.8	Good	Good	20 cc Nu.-Fl.
10							101.0	Good	Good	20 cc H. S.
11	11,750	47	32	20	0	0.6	101.0	Good	Good	20 cc Nu.-Fl.
12							101.0	Good	Good	20 cc Nu.-Fl.
13							101.2	Good	Good	20 cc Nu.-Fl.
14							101.0	Good	Good	
15	11,950	39	40	19	1	1.0	101.6	Good	Fair	20 cc Nu.-Fl.
16							101.2	Good	Good	20 cc Nu.-Fl.
17							101.2	Good	Good	
18	11,750	34	35	27	1	1.0	100.6	Good	Good	Discharged

Without question the dog was placed in the distemper ward and a count, the following morning, of 10,750 total whites and a nuclear index of 0.5 rather definitely confirmed the diagnosis of the evening before.

With a history such as this dog had and the clinical evidence present, together with this type of blood-picture, I feel a diagnosis of canine distemper is very certain. This is another case where the temperature fails to perform in a characteristic manner. This was an older dog, well grown, and in good physical condition. Serum and nucleotide treatment was used in this case, with the dog making a complete recovery.

Nervous canine distemper has been for me the most difficult and unsatisfactory canine disease, considering it from the viewpoint of the veterinarian, the owner and the dog.

Case 5 (table V) was a five-month-old male, Welsh Terrier. The dog was attacked suddenly with convulsions at night, entered a branch office where nembutal was administered and repeated so the dog remained quiet for 48 hours. Microscopical examination of the feces revealed the presence of whipworm and ascarid ova, and tapeworm segments were grossly visible. The parasites were thought to be the etiological factor. The dog did not gain consciousness normally, so a sample of blood was drawn for a count. Referring to table V, we find a blood-picture which, in my experience with dog distemper, commonly accompanies such a syndrome. A 6,350 white count, a nuclear index of 0.8, a temperature of 103.5° F., no appetite and irregular bowel movements constituted the picture.

To begin with, the dog apparently had been sick for some time but had not been observed. He had a white count at least 4,000 below normal and a low nuclear index. I can see nothing but an unfavorable prognosis in such a case. Unless the owner is willing to proceed entirely on "just-a-chance basis," I advise destruction of these dogs. Treatment was started on January 28 with a 15-cc dose of serum and followed by daily 10-cc injections of nucleotide until February 17. The clinical condition, that is, the appetite, bowels, and temperature, made an improvement after February 8 and the dog was discharged on February 21, with a warning that another convulsion might occur at any time and death follow shortly. The animal, to my knowledge, is still alive and enjoys good health, but I do not feel that the life expectancy in these cases is very long.

The balance of this litter, consisting of four other Welsh Terriers, all had distemper at the same time and all made re-

TABLE V—*Nervous canine distemper with hysteria and encephalitis—male Welsh Terrier, five months. (R. B. C., 5,340,000; hemoglobin, 75 per cent; feces, whip, ascarids, tape).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Jan. 28	6,350	38	28	35	0	0.8	103.5	None	None	15 cc H. S.
29							102.8	None	Poor	10 cc Nu.
30							101.8	Loose	Fair	10 cc Nu.
31							101.8	Good	Good	10 cc Nu.
Feb. 1							100.0	Good	Good	10 cc Nu.
2	16,000	20	29	51	0	1.4	100.2	Good	Good	10 cc Nu.
3							102.4	Good	Good	10 cc Nu.
4							100.0	None	Good	10 cc Nu.
5							101.0	Good	Good	10 cc Nu.
6	10,300	36	22	41	0	0.5	101.2	Good	Good	10 cc Nu.
7							102.4	Good	Good	10 cc Nu.
8							101.6	Loose	Good	10 cc Nu.
9							102.0	Good	Good	10 cc Nu.
10							101.2	Good	Fair	10 cc Nu.
11							101.0	Good	Good	10 cc Nu.
12							101.4	Good	Good	10 cc Nu.
13							100.8	Good	Good	10 cc Nu.
14	13,000	42	21	36	0	0.5	101.0	Good	Good	10 cc Nu.
15							101.8	Good	Good	10 cc Nu.
16							101.8	Soft	Good	10 cc Nu.
17							101.6	Good	Good	10 cc Nu.
18							101.6	Good	Good	
19							101.4	Good	Good	
20	10,500	38	33	29	0	0.8	101.6	Good	Good	Discharged

coveries, but none of them developed a 6,300 white count with a low index. I have observed dogs with nervous distemper with high white counts, but I feel that if the disease could be observed throughout its course, that is, from the onset of the virus infection, a leucopenia usually develops. We have had occasion to handle a number of such cases in the past two years and we have had favorable results with about 50 per cent of them. When I say "favorable" results, I mean the dogs are able to be discharged, to remain alive after going home. It is possible with these cases to have them break down a number of months later, so I always explain this possibility to the owner.

I now wish to call to your attention what to me has been one of the most interesting phases of this work. In case 6 (table VI), the blood-picture is prognostic of death and the clinical condition appears favorable. I will use, as an example of this type, the case of a Springer Spaniel, female, two years of age, which, to the owner's knowledge, had never been ill. He had raised the dog from a small puppy. In order to confirm the diagnosis, I will say that the owner first sent in a Gordon Setter with a definite case of canine distemper. I suggested that if he had other dogs, they should be examined also, as the disease was highly infectious.

The following day this Springer arrived. Referring to table VI, we find the stool positive for hookworm but a fair hemoglobin and a lowered red-cell count. The temperature on arrival was 102.4° F. A blood-sample was drawn and simultaneously 20 cc of homologous serum was administered intravenously. Analysis of the count is not alarming. Note a white count of 10,350, immatures 34, segments 56, lymphocytes 14 and a nuclear index of 1.6. The question arose whether the dog was immune. She was in fine coat, good flesh, a splendid specimen of the breed. In view of the contagion I had her placed in isolation and a dose of nucleotide given the next day, followed by a 20-cc dose of serum on the third.

Another count was taken on the fourth and the trend is apparent. The white count has dropped to 8,150 with a nuclear index of 1.1. The dog was placed in the distemper ward and serum and nucleotide therapy continued. I advised the owner that the dog had distemper and that if the count went no lower she would get along nicely. The third count was taken on February 6, with a white count of 4,850 and a nuclear index of 0.4. This presented an alarming aspect, unfavorable prognosis and impending death. The next day the dose of nucleotide was in-

TABLE VI– *Canine distemper with leucopenia—female Springer Spaniel, two years. (R. B. C., 4,860,000; hemoglobin, 89 per cent (15.3 gm); feces, hookworm).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Feb. 1	10,350	34	56	14	0	1.6	102.4	None	None	30 cc H. S.
2							101.4	None	Fair	20 cc Nu.
3							100.2	None	Fair	20 cc N. S.
4	8,150	37	44	19	0	1.1	102.2	None	Good	20 cc Nu.
5							100.8	None	Fair	20 cc Nu.-Fl.
6	4,850	52	24	23	0	0.4	101.0	Good	Good	20 cc Nu.-Fl.
7							100.8	Good	Good	30 cc Nu.-Fl.
8	4,200	37	14	5	1	0.3	100.8	Good	Good	30 cc Nu.-Fl.
9	4,800	51	16	33	0	0.3	100.4	Loose	Poor	30 cc Nu.-Fl.
10	7,100	41	28	30	0	0.6	100.0	Watery	None	20 cc H. S.-Fl.
11	7,450	61	26	13	0	0.4	101.0	Loose	Fair	30 cc Nu.-Fl.
12							101.0	Loose	Poor	30 cc Nu.-Fl.
13	11,400	49	33	18	0	0.6	101.2	Loose	Fair	20 cc Nu.-Fl.
14	12,400	48	31	21	0	0.6	101.4	Loose	Good	20 cc Nu.-Fl.
15	13,300	50	32	30	0	0.6	101.6	Loose	Fair	30 cc Nu.-Fl.
16	14,200	42	22	36	0	0.5	101.4	Loose	Good	30 cc Nu.-Fl.
17							101.4	Loose	Good	30 cc Nu.-Fl.
18	14,600	49	32	19	0	0.6	101.8	Loose	Good	Fluids
19							102.2	Loose	Fair	Fluids
20	11,000	55	34	11	0	0.6	101.8	Watery	Poor	Fluids
21							102.6	Watery	Fair	30 cc Nu.-Fl.
22							102.2	Loose	Fair	20 cc Nu.-Fl.
23	15,900	57	31	12	0	0.5	102.2	Loose	Fair	20 cc Nu.-Fl.
24							102.2	Loose	None	20 cc Nu.-Fl.
25	13,500	49	32	19	0	0.6	102.2	Loose	Fair	Fluids
26							102.4	Loose	None	
27	12,900	56	29	15	0	0.5	102.4	Loose	None	
28							102.2	Loose	Poor	20 cc Nu.-Fl.
Mar. 1	11,250	44	44	11	0	1.0	102.0	Watery	None	20 cc Nu.-Fl.
2							102.0	Loose	Fair	Fluids
3	23,150	46	40	14	0	0.8	102.6	Loose	None	Fluids
4							101.6	Loose	Fair	10 cc Nu.-Fl.
5							102.2	Loose	None	
6	38,100	54	26	20	0	0.4	102.2	Loose	Poor	Fluids
7	33,250	63	26	11	0	0.4	101.0	Loose	None	Discharged

creased to 30 cc in view of the profound leucopenia. The next count, on February 8, shows a still lower white count of 4,200, with a nuclear index of 0.3.

At this point the owner was called to the hospital for conference and he could hardly realize that the animal was in a dangerous condition. On seeing him she jumped about the yard, barked and seemed normal in appearance. The clinic record showed a normal appetite and bowel for two days with a temperature of 100.8° F. The owner said the dog was feeling much better than when she entered the hospital. I explained to him that the dog had every possibility of dying, that treatment in my opinion was hopeless, but that we would be willing to go through with the case if he desired it.

The following day, clinical evidence appeared in the form of loose bowels and a poor appetite. Following the clinical picture from here to its termination, you will note a regularity of the symptoms, that is, loose or watery stools, little if any appetite, and a gradual loss of condition. It is interesting that at no time did this patient show any marked elevation of temperature, 102.4° F. being the maximum reached at any time while under observation. The low point in the blood-picture was reached on February 8. On February 9, the white count began to rise slowly and the index made a little improvement. This incline continued until February 23, with the exception of a slight drop on February 20. The nuclear index, however, is practically stationary between 0.3 and 0.6.

Following the count on February 23, the white count began to decline steadily, reaching 11,250 on March 1. At this point the index jumped to 1.0. The clinical condition was still very unsatisfactory. Then, on March 3, the white count moved up to 23,150, and the nuclear index started down again to 0.8. We lost all hope at this point as such a change usually is indicative of a bacteremia. This was confirmed on March 6 and 7 when the count went up above 30,000 and the index down to 0.4. We advised the owner that treatment was hopeless and suggested destruction. He called at the hospital and said he had decided to take her home and see if she would not regain her strength. She walked out of the hospital and climbed into her car unaided. I was notified by telephone that death followed two days later.

Case 6 has been detailed, as it embodies a number of interesting hypotheses. It emphasized to me the importance of the blood-picture as an aid in diagnosis and prognosis. The unfavorable prognosis given on February 6 was confirmed one month later

TABLE VII—*Canine distemper with leucopenia—male Boston Terrier, eight months. (R. B. C., 5,400,000; hemoglobin, 89 per cent (15.3 gm); feces, negative).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Mar. 9	9,050	30	30	40	0	1.0	102.6	None	Good	
10							101.4	Good	Good	15 cc H. S.
11	8,350	27	22	51	0	0.8	99.6	Good	Good	10 cc Nu.-Fl.
12							102.0	Good	Good	12 cc H. S.
13	7,050	27	26	47	0	0.9	101.4	Good	Good	15 cc Nu.-Fl.
14							101.0	Good	Good	15 cc Nu.-Fl.
15	6,850	37	31	32	0	0.8	101.8	Good	None	15 cc Nu.-Fl.
16							101.6	Loose	Poor	15 cc Nu.-Fl.
17							101.6	Loose	Fair	15 cc Nu.-Fl.
18	8,200	34	27	37	2	0.8	101.8	Loose	Fair	H. S.-Nu.-Fl.
19							101.8	Good	Good	
20							101.0	Good	Good	15 cc Nu.-Fl.
21	9,050	38	24	37	1	0.6	101.8	Good	Good	10 cc Nu.-Fl.
22							101.2	Soft	Good	15 cc Nu.-Fl.
23							101.0	Soft	Good	15 cc Nu.-Fl.
24	12,000	44	33	22	1	0.7	101.8	Soft	Good	15 cc Nu.-Fl.
25							102.0	Soft	None	15 cc Nu.-Fl.
26							102.4	Soft	Fair	10 cc H. S.
27	9,900	50	39	10	1	0.8	102.0	Soft	Good	Discharged

on March 6, when all hope was dismissed. It is evidence that to improve the total white count, without a corresponding improvement in the nuclear index, is valueless. It further substantiates the claim that dogs whose white counts fall to 8,000 and below, with a low nuclear index in cases of canine distemper, are in a dangerous class and deserving of a guarded prognosis. It again points out the unreliability of the temperature curve. A further analysis of this case indicates that, although serum and nucleotide were administered very freely, considering the size of the dog, it had little if any effect.

Such cases, in my opinion, are deserving of biopsy studies of the bone-marrow by a skilled pathologist. I have noticed a rather distinct line of demarcation in treated cases in which the total white counts fall to 5,000 or below with correspondingly low nuclear indices. It appears at present that the mortality in dogs of this group is 100 per cent, regardless of the method of treatment. Dogs whose counts are from 6,000 to 8,000, with a low nuclear index, have a possibility of recovering if serum and nucleotide therapy is used, but even these are deserving of a very guarded prognosis. In the study of the Springer Spaniel, it was indicated that, had the white count remained above 6,000, with a serum and nucleotide treatment, the dog would have had a possibility of recovery.

In order to justify this statement, we shall examine the record of a Boston Terrier (table VII), male, not feeling well for ten or twelve days, with anorexia and intermittently soft stool. The animal was presented on March 9, with a temperature of 102.6° F. He had a slight eye and nasal discharge, and a hacking cough.

Examination of the first blood-picture shows a 9,050 white count with a nuclear index of 1.0. A 15-cc dose of homologous serum was administered intravenously the following morning, at which time the temperature was 101.4° F. The following day, March 11, a second count of 8,350 and nuclear index of 0.8 shows evidence of a further decline, but the clinical condition had improved in that the appetite and bowels were good. The use of nucleotide was started on this date with a 10-cc dose, following by a 12-cc dose of serum the following day. The third count, on March 13, of 7,050 was then below the 8,000 mark and the case took a more serious aspect. The dose of nucleotide was increased to 15 cc and another count on March 15 was still lower, at 6,850, with an index of 0.8. Following this count, the clinical condition began to manifest itself, in that the appetite became poor, the bowels were loose and the temperature remained normal.

TABLE VIII—*Clinical distemper with secondary toxemia—male Dachshund, one year. (R. B. C., 6,210,000; hemoglobin, 89 per cent (15.3 gm); feces, negative).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Feb. 13	8,550	55	41	4	0	0.7	102.6			20 cc H. S.
14							101.8	Loose	Fair	10 cc Nu.-Fl.
15	8,450	44	34	21	0	0.7	101.6	Good	Fair	10 cc Nu.-Fl.
16							101.6	Good	Good	15 cc Nu.
17	8,800	38	30	30	2	0.8	101.0	Good	Good	20 cc Nu.
18							100.8	Good	Good	20 cc Nu.
19							102.0	Good	Good	20 cc Nu.
20	14,050	37	46	16	0	1.2	101.2	Soft	Good	20 cc Nu.
21							102.4	Loose	Good	20 cc Nu.
22							101.6	Loose	Good	
23	9,550	38	52	11	0	1.3	101.4	Loose	Good	
24							101.6	Good	Good	
25							101.0	Good	Good	
26							101.0	Good	Good	
27	16,850	39	48	8	1	1.3	102.0	Good	Good	
28							101.2	Good	Good	Discharged

Serum and nucleotide were administered jointly on March 18 at which time the total white count had moved up to 8,200, but the index remained the same. By March 24 the count was up to 12,000 but the index was not improving. The last count available on this patient showed a 9,900 white count with an index of 0.8 and the dog was removed from the hospital by the owner with a guarded prognosis. The dog was presented again in the evening, three days later, with a report that the appetite was returning and the bowels were good. The dog had a normal temperature and was showing clinical improvement.

It is unfortunate that we are not always able to follow these cases to their termination, but this is not possible in handling clinical cases. A later report by phone indicated that the dog seemed perfectly normal and, to my knowledge, is alive and well at the present time. It must be said, however, that this dog did not develop a profound leucopenia with an excessively low nuclear index and the lymphocyte count was favorable up until the time of discharge. It is my opinion that cases of this type run a very high mortality unless promptly treated with homologous serum and nucleotide.

Proceeding to another clinical case (table VIII), but of a little different type, we have for consideration a long-haired Dachshund, male, one year old. This dog had been under veterinary supervision for two or three weeks prior to presentation. Definite clinical signs were in evidence, with emaciation, poor coat and congested eyes. The respiration was forced and rapid, the bowels loose and fetid, and there was little if any nasal discharge. This dog was toxic and very sick. The temperature was slightly elevated and the dog was refusing food. The question of diagnosis in such a case was, without a doubt, simple; but, what was the prognosis?

Now, returning to table VIII, we note a total white count of 8,550, 55 immatures, 41 segments and 4 lymphocytes, with a nuclear index of 0.7; a temperature of 102.6° F., loose, watery bowels, and refusing food. Considering the entire picture with a closely bred, show-type, hound dog, the prognosis certainly looked unfavorable. I wish to emphasize at this point that, at this state of the disease, with a white count of 8,000 and an index of 0.7, such a blood-picture is an exceedingly dangerous one. Treatment was instituted by the intravaneous injection of a 20-cc dose of homologous serum. I feel that in a case such as this, serum is of some value but it does not produce the results noted when injected at an earlier period of the disease. We followed

TABLE IX—*Canine distemper complicated with marked secondary infections—male Collie, two years. (R. B. C., 4,050,000; hemoglobin, 79 per cent (13.6 gm); feces, negative).*

DATE	W. B. C.	IM.	SEG.	LYM.	MON.	N. I.	TEMP. (F.°)	BOWEL	APPETITE	TREATMENT
Feb. 27	24,550	61	26	13	0	0.4	105.0	None	None	
28							104.2	None	Poor	20 cc Nu.-Fl.
Mar. 1	35,450	56	34	10	0	0.6	102.8	None	Fair	30 cc Nu.-Fl.
2							101.0	Good	Fair	Fluids
3	28,650	43	45	12	0	1.0	100.0	None	Fair	Fluids
4							100.0	Good	Fair	Fluids
5							100.0	None	Good	Fluids
6	18,350	33	38	27	0	1.1	101.0	None	Good	Fluids
7							100.2	Good	Good	Fluids
8	13,050	40	41	18	0	1.0	101.8	Good	Good	Discharged
May 12										20 cc H. S.
13	19,050	50	43	7	0	0.8	104.2	None	Poor	20 cc Nu.-Fl.
14							101.4	Soft	Fair	30 cc Nu.-Fl.
15	20,050	46	37	15	0	0.8	101.6	None	Fair	30 cc Nu.-Fl.
16							101.4	Good	Poor	Fluids
17							101.8	Good	Good	Fluids
18							101.0	Good	Good	Fluids
19							101.4	Good	Fair	Fluids
20	17,000	41	31	26	1	0.7	101.6	Good	Fair	20 cc Nu.-Fl.
21							101.2	Good	Good	
22	16,200	37	38	24	0	1.0	100.8	Good	Good	Discharged

this single injection of serum with daily doses of nucleotide intravenously.

On February 20, the blood-picture showed 14,050 total whites with a nuclear index of 1.2. We were not concerned with a 9,550 white count on February 23, as we had a slowly improving index of 1.3. I must again call to your attention the fact that this dog at no time carried an excessively high temperature. The favorable improvement in the appetite and bowels, with a normal temperature after February 23, is also some evidence of a recuperating patient. This dog was later examined by another veterinarian who reported him in a good state of health.

We shall now consider briefly the type of case (table IX) with which we are all familiar. This was a blue merle Collie, male, two years old, very well bred, and had been under veterinary supervision for two or three weeks. When presented on February 27, the animal was hardly able to walk into the clinic. He had pleuritis, pneumonia, complete anorexia, bowel stasis, and was very toxic, weak and sick. The temperature was 105° F. The blood showed a 24,550 white count, 61 immatures, 13 lymphocytes, with a nuclear index of 0.4. My prognosis on a case such as this usually is quite unfavorable. The man wished the animal treated, so with this understanding we proceeded.

The initial treatment consisted of a 20-cc dose of homologous serum, some saline and glucose, and a pneumonia jacket. The white count went up to 35,450 on March 1, with a reduction of the immatures, increase in the segments, and an improvement in the nuclear index of 0.6. Following this count the next reports, on March 3, 6 and 8, showed declining total white counts with a slow upward movement in the nuclear index. The dog started to eat, the bowels became normal and he showed a marked clinical improvement. The dog was removed from the hospital by the owner contrary to our advice. He remained home until May 12, when he was returned with a temperature of 104.2° F. The white count was 19,050 with a nuclear index of 0.8. We again treated the dog for a period of ten days, after which he was removed.

The second installment in the hospital was similar to the first, except that the white count did not go quite so high or the index so low. The animal was again removed against our advice and, on the last report, was making only a lingering recovery. I am showing this case, not from a therapeutic angle but from the standpoint of diagnosis and prognosis. It is not difficult to convince the owner of a dog such as this that the animal is seriously

sick as clinical symptoms are very much in evidence. From the veterinarian's point of view, however, to bring about a favorable recovery is much more difficult. It must be remembered that the resistance in such a case has been seriously damaged by the filtrable virus and now with an overwhelming invasion of secondary bacteria and their associated toxins the prognosis is necessarily a very guarded one.

SUMMARY

Following an analysis of these specimen reports, it would seem that dogs presented for veterinary attention and possibly afflicted with canine distemper could be divided into the following groups, depending upon the type of blood-picture and clinical evidence present.

I. *Early virus:* Dogs in this group show little if any clinical evidence of canine distemper, but may have an increased white blood count and lowered nuclear index. The clinically normal dog presented for immunization and found to have an abnormal blood-picture frequently belongs to this group.

II. *Early clinical canine distemper:* This might also be considered a virus group with little if any secondary infections. These cases frequently show an elevation of temperature although not constantly. A slight coryza, disturbed appetite, chills and the early symptoms of a common cold are frequently observed. The blood-picture of this group is somewhat variable but commonly shows an elevation of the white count and a lowering of the nuclear index. In very virulent infections, the white count may be normal or subnormal with a correspondingly low index. Dogs of this group must be observed very carefully to avoid error in diagnosis.

III. *Clinical canine distemper:* These are the cases commonly presented at a clinic, as the owner can usually see definite symptoms. The temperature often fluctuates between 102 and 104° F., with days when the temperature is normal. The clinical symptoms are variable. They often have eye, nose and throat symptoms. The appetite may be reduced with disturbed bowel functions which are usually soft, loose and sometimes hemorrhagic and watery. The blood-picture will vary somewhat, but the white count is frequently normal or subnormal with a low index.

IV. *Canine distemper with leucopenia:* This group of dogs is characterized by no definite syndrome. In some cases meningitis and encephalitis are apparent much earlier than in others. It has been my experience that the majority of these cases do not show the marked eye, nose and throat symptoms and they are

sometimes described by the kennel man as dogs with "dry distemper." This is the high mortality group. In this group, the ones which survive are commonly afflicted with chorea, paralysis, or physical impairments. The blood-picture is usually quite diagnostic with a subnormal white count between 3,000 and 8,000 and a low nuclear index from 0.2 to 1.0. The onset of sepsis will, of course, alter the picture.

V. *Canine distemper with secondary complications:* Dogs in this group are closely allied to those of group IV. The symptom complex, however, is frequently somewhat different. These dogs are always visibly very ill with pleuritis, pneumonia, heavy discharges from the eyes and nose, disturbed bowels, partial or a complete anorexia, toxic, and very sick. The blood-picture as a rule is quite typical. It usually shows a "shift to the left," with leucophilia and a low nuclear index.*

Discussion

In examining the representatives of these groups individually, they may be considered atypical in certain respects. First, they do not conform with the temperature curve commonly diagnostic of this disease. Second, the clinical manifestations are not constant. The appetite and bowels may be normal and the dog may have little if any eye, nose and throat symptoms. Last, these reports are gleaned from daily practice. The entire picture has been altered by the application of therapeutic measures. Such cases have received the contagion from a street virus highly variable in virulence and dosage. It is not possible, therefore, to compare in detail these records with published reports based upon experimentally produced canine distemper.

It must also be kept constantly in mind that, in the application of the blood picture as an aid in the diagnosis of this disease, an infectious shift is also possible in diseases other than canine distemper. The veterinarian must therefore apply sound principles of diagnosis in every case and not form immature opinions from a single blood-picture.

Thanks to the efficient investigation of Laidlaw and Dunkin[1-5] and many American institutions, the present prophylaxis and treatment of canine distemper have been greatly enhanced. In homologous serum we have a valuable therapeutic measure. It is especially efficient when used in large doses in controlling the

*All the differential cell counts in this report are expressed in percentages, based on the classification of 100 cells. We have more recently used the technic described by Kolmer and Boerner.[11] The reporting of each individual cell in total numbers is preferred, *e. g.*, white blood count 10,000, of which 50 per cent are mature neutrophiles, would indicate 5,000 of this type per cmm.

filtrable virus of this disease. It is also a valuable prophylactic for puppies two or three months of age and susceptible hospital patients. The efficiency of this product, however, in my experience is considerably diminished after the onset of definite clinical symptoms which commonly follow the degradations of the virus. It is here that further assistance becomes necessary. In view of this need, we started the administration of nucleotide.

I do not wish to convey the impression that serum and nucleotide therapy is a positive cure for all cases of canine distemper. I do feel, however, that nucleotide has proved a real benefit to us in a great many cases and has been responsible for decreasing the mortality rate in dogs which we have treated. In compiling the results obtained at our institution from September, 1932, to August, 1933, I find we observed about 300 dogs in which a diagnosis of canine distemper was made. Of these dogs, 150 were ward cases and the balance ambulatory or office calls. The mortality in the entire group was 15 or 16 dogs.

We treat very few developed cases of this disease in the ambulatory clinic. Dogs treated in this clinic are usually kennel cases. In such instances, the most malignant ones are moved to the hospital immediately and the balance treated on the premises. These figures do not include dogs destroyed on the day of entry or the day following which were in a hopeless condition and to which no treatment was given. This is the most encouraging result we have been able to obtain in the hospitalization and treatment of canine distemper. We attribute what we feel was a more successful year to the assistance of laboratory diagnosis and the prompt application of homologous serum and nucleotide together with a rich vitamin diet and sanitary hospitalization.

I will now attempt to give some idea of the equipment we use in carrying on this work. It is difficult to picture an institution that would be ideal in every respect for the hospitalization of canine distemper. In our case we use one room as a general clinic room and try to avoid handling distemper patients there. We house distemper cases in another ward and exercise the animals in the adjoining yard. All distemper cases are treated in this ward. We do not have a separate clinic room for dogs suffering with this disease, which would be an asset. Our general hospital cases use the other yards. An entirely separate building would be of distinct advantage, but materially increases the expense of handling the disease. With this arrangement, and prior to the adoption of laboratory assistance in diagnosis, we encountered some difficulty with breaks in non-infectious wards. This has

been decidedly minimized the past two years and we infrequently find it necessary to move surgical and clinical cases to the distemper ward.

We rarely accept a young patient in our clinic without emphasizing to the owner the necessity of conducting a physical examination, including a laboratory report on the blood and feces. This is requested to determine the actual condition of the dog. It requires but a few hours and frequently we, as well as the owner, have been spared an unfortunate situation.

The control of this infection in a veterinary hospital is a real administrative problem. In my opinion, the unfortunate reputation so often delegated to the veterinary hospital by the public can be reclaimed and maintained by the application of scientific methods of diagnosis, sanitation and intelligent therapeutics.

REFERENCES

[1]Dunkin, G. W., and Laidlaw, P. P.: Studies on dog distemper. I. Dog distemper in the ferret. Jour. Comp. Path. & Therap., xxxix (1926), 3, p. 201.
[2]Dunkin, G. W., and Laidlaw, P. P.: Studies on dog distemper. II. Experimental distemper in the dog. Jour. Comp. Path. & Therap., xxxix (1926), 3, p. 213.
[3]Laidlaw, P. P., and Dunkin, G. W.: Studies on dog distemper. III. The nature of the virus. Jour. Comp. Path. & Therap., xxxix (1926), 3, p. 222.
[4]Laidlaw, P. P., and Dunkin, G. W.: Report of field investigations of canine distemper. Vet. Med., xxiv (1929), 5, p. 210.
[5]Dunkin, G. W., and Laidlaw, P. P.: Some further observations on dog distemper. I. The durability of the immunity following vaccine and virus administration. Jour. A. V. M. A., lxxviii (1931), n. s. 31 (4), pp. 545-551.
[6]Jackson, Parker, Rinehard, and Taylor: The treatment of malignant neutropenia with pentose nucleotides. Jour. Amer. Med. Asso., xcvii (1931), pp. 1436-1440.
[7]Jackson, Parker, and Taylor: The nucleotide therapy of agranulocytic angina, malignant neutropenia and allied conditions. Amer. Jour. Med. Sci., clxxxiv (1932), 3, p. 297.
[8]Morris, M. L.: Nucleotide K-96 in the treatment of canine distemper. No. Amer. Vet., xiv (1933), 5, pp. 32-51.
[9]Schilling, V.: Blood Picture and Its Clinical Use. (Mosby Co., Saint Louis, Mo.)
[10]Piney, A.: Recent Advances in Haematology. (P. Blakiston's Son & Co., Philadelphia, Pa.)
[11]Kolmer, J. A., and Boerner, F.: Approved Laboratory Technic. (D. Appleton & Co., New York, N. Y., 1931.)
[12]Gerard, J. H., and Boerner, F.: The significance of "shift to the left" in differential leucocyte counts and the nuclear index as a means for interpreting and recording (a) the nuclear index of normal blood and the influence of age; (b) the nuclear index in disease. Jour. Lab. & Clin. Med., xvi (1930), 3.
[13]Farley, D. L., St. Clair, H., and Reisinger, J. A.: The normal filament and nonfilament polymorphonuclear neutrophile count: Its practical value as a diagnostic aid. Amer. Jour. Med. Sci., clxxx (1930), 3, p. 336.

Kansas Veterinarians Meet in Topeka

A joint meeting of the Northeast Kansas Veterinary Medical Society and a special called meeting of the Kansas Veterinary Medical Association was held at the Hotel Jayhawk, Topeka, June 1, 1934. The 55 veterinarians in attendance reported an enjoyable time.

E. H. L.

THE REACTION OF SWINE FOLLOWING EXPERIMENTAL INOCULATION OF A PATHOGENIC STRAIN OF BRUCELLA ABORTUS OF PORCINE ORIGIN*

By WILLIAM H. FELDMAN *and* CARL OLSON, JR.
Division of Experimental Medicine
The Mayo Foundation, Rochester, Minn.

Recently we have published[1] the results of a study of spontaneous spondylitis of swine, in cases of which bacteria of the Brucella group were isolated in many instances. The number of strains of *Brucella abortus suis* obtained seemed sufficient for this bacterium to be of etiologic significance in the pathogenesis of the disease.

The condition was characterized by the occurrence of localized abscesses, with apparent predilection for the body of the vertebral structures. All of the bacterial strains obtained, with the exception of one, proved pathogenic for guinea pigs and in some instances for rabbits. Consequently, it seemed pertinent to ascertain if an organism obtained from a typical lesion of spontaneous spondylitis was capable of inducing a specific agglutinative response in swine exposed experimentally, and if significant lesions would follow artificial inoculation.

METHODS

Eight swine of the Duroc Jersey breed, approximately seven months of age, were given intradermal injections of avian and mammalian tuberculin, and all failed to reveal sensitivity to either product. Likewise, the blood of none of the animals reacted positively to the agglutination test for Brucella infections in dilutions of 1:25, 1:50 and 1:100.

The bacterial culture used to inoculate the respective swine had been isolated by direct culture 60 days previously, from a lesion in one of the lumbar vertebrae of a hog approximately one year of age. The animal apparently was in good health at the time of slaughter, and the lesions were found by the veterinary inspector during the routine postmortem examination. The lesion consisted of necrotic cavitation in the epiphysis of one of the lumbar vertebrae, which extended laterally into the soft tissues on one side and caused bulging of the involved parts. When opened, the abscess of the soft parts was found to consist of a semi-caseous substance, well encapsulated from the surround-

*Received for publication, November 16, 1933.

ing tissue, but connected with the lesion in the vertebra by an irregular, tortuous channel, also filled with necrotic material.

From the lesion in the vertebra an organism of the Brucella group was obtained in pure culture, and an emulsion prepared from the necrotic content of the lesion induced specific infection in rabbits and guinea pigs. Blood obtained from these animals eight weeks subsequent to inoculation contained agglutinins for a polyvalent antigen prepared from several strains of the Brucella organism. At necropsy, the pathologic characteristics of a Brucella infection were observed, and the organism was obtained in pure culture from the spleens of the respective experimentally inoculated animals.

Dr. K. F. Meyer, University of California, and Dr. I. Forest Huddleson, Michigan State College, were supplied subcultures of the strain originally isolated directly from the vertebral lesion of the spontaneously infected hog and each considered the organism to belong to the Brucella group and to possess biologic characteristics typical of the porcine variety of this genus.

A subculture of the organism obtained direct from the original vertebral lesion was grown at 37.5°C. for ten days, in glucose-brain broth, and was used for inoculation of the eight swine as follows: Swine 1 and 2, females: each received subcutaneously 1 cc of the bacterial inoculum. Swine 3 and 4, females: 0.25 cc of the culture was placed in contact with the mucosa of the conjunctiva of the left eye of each animal. Swine 5 and 6, males: given intravenous injection of 0.5 cc of the bacterial culture. Swine 7 and 8, one male and one female: each received orally a gelatin capsule containing 1.5 cc of the bacterial inoculum.

Each group of two animals was placed in a separate pen and entirely apart from quarters previously occupied by other hogs. Blood was secured at frequent intervals for agglutination tests, and 319 days subsequent to experimental exposure, the respective animals were killed for necropsy.

Agglutination Tests

The antigen used for determination of specific agglutinins in the blood of the respective swine was a polyvalent preparation obtained from Dr. C. P. Fitch, University of Minnesota. The presence of agglutinins was determined by the so-called tube method. Sixteen different specimens of blood were secured from each animal; the first blood was obtained one week after exposure to the infective bacteria. The last blood taken for agglutination test was obtained at the time the animals were killed for ne-

cropsy. Compilation of the results obtained, expressed in the highest titre for each test specimen, is summarized in table I.

Specific agglutinins were produced in the blood of each of the eight animals exposed to the infective inoculum. The degree of infection, as measured by the agglutination test, varied with the method of inoculation and to some extent with the individual animal. Agglutinins became demonstrable earliest in the blood of those animals which received the bacterial culture intravenously and subcutaneously, and appeared latest in the two swine which received the culture by mouth. The two animals that were given inoculation into the membranes of the eye differed markedly in the appearance of agglutinins. Swine 3 gave a titre of 1:50 two weeks after exposure to the infection; this titre was evident also at the next test, eight days later. This was followed by three negative tests, after which an agglutinative titre of 1:25 was obtained for several tests. The tests then became negative, again, however,

TABLE I—*Maximal agglutinative titres obtained from swine inoculated with a pathologic strain of the Brucella group of bacteria isolated from the lesion of spondylitis in a hog.**

Days After Inoculation	Animals and Route of Inoculation							
	Subcutaneous		Conjunctiva		Intravenous		Oral	
	1	2	3	4	5	6	7	8
8	1:50	1:50	—	—	1:50	1:50	—	—
14	1:100	1:50	1:50	—	1:400	1:200	—	—
22	1:50	—	1:50	—	1:100	1:100	—	1:100
29	1:100	—	—	—	1:100	1:100	1:50	1:50
35	1:100	—	—	1:100	1:50	1:50	1:50	1:50
50	1:100	1:100	—	1:50	1:100	1:200	1:25	—
64	1:50	1:100	1:25	1:50	1:100	1:200	1:25	1:50P
80	1:100	1:200	1:25	1:100	1:100	1:200	1:25	1:50
98	1:50	1:100	1:25	1:200	1:50	1:400	1:50	1:25
112	1:50	1:200	1:25P	1:200	1:100	1:200	1:50	1:25
126	1:25	1:100	—	1:200	1:50	1:400	1:50	1:25P
146	1:25	1:50	—	1:200	1:50	1:400	1:50	—
168	1:12	1:25	—	0	1:25	1:200	1:50	1:12P
236	1:12	1:25	1:25P	1:100	1:25	1:100	1:50	1:12P
290	1:12	1:25	1:25P	1:50	1:25	1:50	0	—
319	1:12	1:25	1:50	1:50	1:12	1:50	1:50	1:12

*The first test was made one week after exposure to infection.
— = Test negative.
P = Agglutination in the stated dilution incomplete.
O = Data not available due to blood specimens becoming hemolyzed.
Note: The lowest dilution used in the first five tests was 1:50. For the next seven tests, including the test on the 50th day, the lowest dilution was 1:25. Beginning with the test on the 168th day, the lowest dilution was 1:12.

to reveal partial agglutination at 1:25 on two occasions, and on the day the animal was killed a titre of 1:50 was obtained. The mate of this hog, although requiring more time to develop agglutinins, maintained a fairly consistent titre for a considerable period of the experiment, and in no instance was a negative test obtained once the agglutinins became evident.

The hogs inoculated subcutaneously revealed agglutinins after one week, yet the ultimate titre obtained from the blood of one was somewhat higher than the titre demonstrated in the blood of the other.

In the two swine which received the infection by ingestion apparently a high agglutinative titre never developed. This is in marked contrast to those swine which were given intravenous inoculation; in these two animals a titre of 1:50 developed within one week after they had received the inoculation, and the titre became higher than that of any of the other animals in the series. There was, however, a difference between agglutinative responses of the two animals mentioned. With the exception of the first five tests, the agglutinative response of one of the swine was consistently lower than that of the other. In the latter animal, titres that were in excess of those obtained from any of the other animals eventually developed and were maintained for a considerable period of the experiment.

Agglutinins, when once produced, remained consistently present, although in gradually diminishing degree, in practically all of the animals except two (swine 2 and 3). Periods of variable duration occurred, when agglutinins were not demonstrated in blood-serums from these two animals. The explanation of this apparent disappearance and eventual reappearance of agglutinins is not evident. Although it may indicate a temporary let-down of that portion of the protective mechanism represented by agglutinins, the possibility of recovery and reinfection from the other infected animal that occupied the same quarters, and the consequent reappearance of agglutinins, should be considered. Swine 2 and 3 were confined in different pens, but the pen-mates of each of these animals constituted possible sources of reinfection, particularly in view of the fact that agglutinins reappeared in the earlier phases of the experiment, when the infective state was presumably most active. However, proof of either of these hypotheses is absent.

Necropsy of Inoculated Swine

At the time of slaughter, all of the swine were carefully examined for morphologic evidence of infection. Particularly were

we interested in the possibility of lesions of spondylitis having developed, and in order to determine whether they had developed, the vertebral column was split longitudinally, so that the body of each vertebral segment might be examined. With the exception of lesions involving both kidneys of swine 7, no pathologic changes were observed in any of the animals. The cortical portions of both kidneys of swine 7 contained grayish-white abscesses, measuring approximately 4 cm in diameter. Cultures were made from the creamy, purulent content of each lesion, and a pure culture of a Gram-negative, spore-forming bacillus was obtained from each. Guinea pigs were also inoculated with portions of the material from the abscess. These were kept under observation for 50 days, and were negative for infection with Brucella when killed for necropsy.

ATTEMPTS TO OBTAIN BRUCELLA ABORTUS FROM SWINE SUBJECTED TO NECROPSY

When necropsy was done it seemed desirable to secure tissues from the respective swine to determine if the infective organism were present. Previous observations[2] had confirmed the findings of Johnson and Huddleson[3] that the spleens of hogs spontaneously infected with bacteria of the Brucella group were the organs most likely to harbor the bacteria. Since this seems to obtain also in guinea pigs experimentally infected, approximately 8 gm of splenic tissue was secured from each hog and emulsions were prepared. Each emulsion was used for subcutaneous injection of two guinea pigs. After a lapse of 49 days, the guinea pigs were killed for necropsy, and blood was secured for agglutination tests. All of the guinea pigs were without gross evidence of disease. The spleen of each guinea pig was removed in a sterile manner and was preserved in the refrigerator for the purpose of making cultures if the results of the agglutination tests revealed evidence of infection.

The respective blood-serums were added to a polyvalent Brucella antigen in dilutions of 1:25, 1:50 and 1:100, and in no instance did agglutination occur. The failure to demonstrate specific agglutinins in any of the serums precluded the necessity of attempting isolation, from the different spleens, of bacteria of the Brucella group.

INOCULATION OF PREGNANT SOW

Some months after inoculation of the eight swine previously mentioned, an opportunity was had to determine if the infective bacterium would exert a pathogenic effect on a pregnant sow.

When given the injection, the animal was considered to be perhaps three weeks from the normal termination of pregnancy, although the exact date of conception was not known. Before the animal was inoculated, blood was obtained for an agglutination test, and agglutinins for an antigen prepared from organisms of the Brucella group were not demonstrated. One cc of a 48-hour culture in dextrose-brain broth of the same strain of Brucella that was used for inoculation of the eight other swine was injected directly into the heart. One week later, an agglutinative titre of 1:200 was obtained, and eleven days subsequent to exposure four dead fetuses were born and another, also dead, was delivered manually. Twenty-seven days after the sow received the bacterial suspension, blood from the animal was found to possess an agglutination titre of 1:800. The titre had dropped to 1:100 after 127 days following exposure, and now, after approximately eight months, it is 1:50.

Heart-blood secured from the respective fetuses that were born dead failed to reveal agglutinins in the presence of an appropriate antigen, and organisms of the Brucella group could not be isolated from the spleens. Cultures were attempted also from remains of the placental membranes removed from the vagina of the sow, but contamination made identification of specific bacteria impossible. Guinea pigs were inoculated with portions of an emulsion prepared from the placental tissues, but the results were negative.

Additional Note on the Pathogenicity of the Bacterial Culture Used to Inoculate the Swine

Subcultures of the Brucella bacterium used in this experiment have been utilized in an additional study pertaining to the pathogenicity and tenacity of the organism.* This work was started some months after the swine had been inoculated, and is being continued. It seems important to mention this, since we see no evidence of diminution of the pathogenicity of the strain after its artificial cultivation for more than 15 months. In other words, although the organism failed to induce morphologic evidence of disease following its introduction into an animal of the same species from which it originally had been obtained, it has continued to be pathogenic for guinea pigs months after the swine had been exposed.

However, it is our opinion that the pathogenicity of this and other strains obtained from swine with spondylitis may represent bacteria of reduced virulence, since the pathologic changes in-

*To be reported in detail in another paper.

duced in guinea pigs are seldom as pronounced as those generally reported following injection of these animals with bacteria of the genus Brucella, obtained from other morbid processes of infected swine. Agglutinins are always produced, and in some animals a rather high titre (1:6,400) may be observed, but the morphologic changes are usually limited to hyperplasia of the spleen, frequently without focal lesions, and epididymitis. Foci are occasionally seen in the liver, but we have never encountered involvement of the joints. Not a few of the guinea pigs inoculated with the cultures obtained from various lesions in cases of spondylitis have been practically devoid of significant gross lesions when killed eight weeks after inoculation, yet specific agglutinins can be demonstrated in the blood, and the organisms can be isolated from the spleen.

COMMENT

Few reports dealing with the pathogenicity of strains of bacteria of the Brucella group obtained from porcine sources have been published since Traum[4] isolated and identified the organism in 1914. Résumé of some of the more significant work is helpful in understanding the behavior of this organism in what may be presumed to be its natural host.

Good and Smith,[5] using an organism that had been obtained from an aborting sow, were able to cause abortion of two pregnant sows. The bacteria were injected intravenously, and agglutinins were demonstrated in the blood of each animal. The infecting organism was recovered from a fetus of each of the infected sows.

Hayes[6] failed to isolate the organism from the testes of 18 boars, 17 of which had been artificially infected, whereas one harbored a spontaneous infection. Hayes did, however, succeed in isolating *Br. abortus suis* from the mammary gland of one sow that had been artificially infected by feeding. Hayes was also unable to obtain the organism from various tissues of two barrows that were presumed to harbor the infection on the basis of a positive agglutinative titre. He also attempted to cause abortion of six pregnant sows by experimental inoculation. Three of the animals were given intravenous injection, and three received the infective agent by mouth. Only one of the animals aborted.

In a later report[7] in collaboration with Howarth, Hayes reported the intravenous injection of four pregnant sows with a culture of *Br. abortus* of porcine origin. Although the titre of the blood remained unusually high, and the infective organism was isolated from the tissues of three of the sows, abortion did not occur.

Cotton,[8] working on the pathogenicity of *Br. abortus* of porcine origin, failed to induce a permanent infection in boars by intravenous injection, but injection of the organism directly into the testes established an infection of lasting duration. Infection of pregnant sows followed feeding of the organism.

Weeter[9] fed to a sow, on the 48th day of pregnancy, a suspension of *Br. abortus suis* but abortion did not subsequently occur. At the termination of the normal period of gestation, there were farrowed five healthy pigs and one macerated fetus, from the tissues of which the infecting organism could not be isolated. The gradual diminution of the agglutinative titre indicates that the infection was eliminated, and this was further emphasized by failure to recover the organism from the tissues of the inoculated sow, at her death six months after the last exposure. Weeter also mentioned two gilts that were repeatedly exposed to the infection by feeding. The animals gave slight and transitory agglutinative responses, whereas in a boar that was similarly exposed demonstrable agglutinins failed to develop.

Starr[10] was able to demonstrate agglutinins in low titre in swine following exposure to *Br. abortus* derived from swine. The infecting bacteria were placed in contact with the skin and mucous membranes. Agglutinins appeared in the blood approximately five weeks subsequent to exposure. The infection was transitory, and tended to disappear in about two months.

Cotton and Buck[11] subjected twelve pregnant sows and two boars, each to a single exposure to infection with Brucella by placing in both eyes of each animal a suspension of *Br. abortus* of porcine origin. All but one of the animals became infected, as was revealed by positive agglutination tests, and likewise the organism was recovered from the blood-stream of all but one animal, by inoculation of guinea pigs. Although some abortions occurred, the authors concluded that exposure of pregnant sows to *Br. abortus* of porcine origin, by way of the conjunctiva, did not regularly result in abortion.

The results of our attempts to induce infection in swine by an organism originally obtained from a porcine source are in general not dissimilar to those of other workers. Although we did not succeed in establishing focal lesions that were demonstrable at necropsy, nevertheless if one assumes that a significant agglutinative titre represents a state of infection, then all of the swine in our series became infected. The significance of the presence of specific agglutinins in this instance may well be the subject of speculation. Because specific agglutinins were produced

in the respective swine it does not necessarily follow that infection was established, since the agglutinogenic ability of nonviable bacteria is well known. Our failure to secure the infective organism from the tissues of the respective swine may have been due to one of two reasons. Either the organisms were absent from the particular tissue examined, and were present elsewhere in the body, or the infection gradually had been eliminated, leaving no morphologic evidence of its former residence.

We believe that the infection induced was in every animal a transitory one, and that the presence of agglutinins represented a reaction that was gradually but definitely diminishing. We believe, further, that organisms of the Brucella group may be harbored in tissues of the body and not provoke recognizable morphologic changes. The duration of the experiment was sufficient to permit of recovery from an infection that might have been established soon after exposure to the infective bacteria, and the resultant lesions gradually and finally might have been obliterated by reparative processes. The technical difficulties of conclusively proving the presence or absence of *Br. abortus* in the tissues of swine, or, in fact, of any animal, are formidable. To utilize, for inoculation of guinea pigs, representative portions of all tissues of an experiment animal, is obviously impractical and, for most purposes, hardly necessary. In view of the results obtained previously, as a consequence of the procedure followed, we feel reasonably certain that viable, virulent bacteria of the Brucella group were not present in the respective animals, and had the animals been permitted to live for an indefinite length of time it is not unlikely that the agglutinative titres eventually would have disappeared.

The inability of the bacteria to promote morphologic changes of sufficient degree to insure the presence of lesions at necropsy would indicate the possession by swine of resistance, of considerable degree, to the particular strain of bacteria used. It seems somewhat curious that a microörganism originally obtained from a well-established lesion of a hog, and which has consistently exhibited ability to produce the pathologic change of infection with Brucella in guinea pigs should be relatively innocuous to other swine. There undoubtedly are many factors that are essential to the production of a state of disease which is manifested by the occurrence of lesions and objective symptoms. It is not unlikely that differences in the virulence of various strains of the organism are of considerable significance in explaining this phase of the problem, although the susceptibility of different species of

animals, and of different individuals of the same species, are worthy of consideration.

When it is considered that the incidence of occurrence of spontaneous spondylitis associated with bacteria of the Brucella group, in one series that we compiled, was approximately one in every 6,000 swine, it is perhaps not remarkable that we failed to induce the specific lesions in the relatively small group of animals inoculated. The occurrence of infection with Brucella among swine, as indicated by serologic tests, varies greatly, but the disease is not infrequent.* The infrequency with which the disease localizes spontaneously in the bones of the vertebral column, even though the infection is not uncommon in swine, makes our failure to induce spondylitis less surprising.

In contrast to the results we obtained following experimental exposure of swine to infection with Brucella of porcine origin are the observations of Makkawejski, Kardadinowskaja and Micheew.[13] These authors gave a clinical and pathologic description of spontaneous infections of swine with Brucella as the disease occurs in so-called White Russia. In addition to abortion, the outstanding manifestations of the disease, as observed by these writers, consisted of orchitis, arthritis, impotence and lameness. Abscesses were noted frequently in the submaxillary cervical, inguinal and popliteal lymph-nodes. Pyemic arthritis, and other lesions of the legs of swine associated with lameness were reported also by James and Graham[14] in several swine in Illinois.

A résumé of the observations which have been recorded during the relatively short period since the specific character of infections of swine with Brucella was established enables us to outline briefly the salient features that characterize the pathology of this disease. 1. The disease is widely distributed over the United States and much of Europe. 2. The infection is not restricted in its anatomic predilection. It may affect the tissues of the genital tract but is frequently extra-genital in its manifestations. The bones of the spinal column and the joints of the legs are not uncommonly involved. 3. Anatomic alterations may or may not be observed in tissues which eventually yield the specific bacterium by laboratory methods. 4. The severity of the infection varies within wide limits. 5. There seems to be a marked difference in the virulence of the etiologic agent as it occurs in different animals and in different parts of the world. 6. The disease varies in severity and recovery probably is fre-

*In one group of 3,975 hogs slaughtered in Michigan, Huddleson, Johnson and Hamann[12] found agglutinins in 7.74 per cent.

quent. It is enzoötic and is apparently far more prevalent than the occurrence of abortions among swine would indicate.

SUMMARY AND CONCLUSIONS

With the use of a pathogenic strain of *Brucella abortus* of porcine origin, originally isolated from a lesion of spondylitis, attempts were made to induce lesions in eight swine. The animals were separated into four lots of two animals each and were inoculated as follows: two intravenously, two by mouth, two subcutaneously, and two by exposure through the conjunctiva. Although infection presumably occurred, since agglutinins were demonstrable in the blood-serum of all the animals, lesions could not be demonstrated when the animals were examined at necropsy, 319 days subsequent to exposure. Furthermore, attempts to isolate *Br. abortus*, by injecting into guinea pigs emulsions prepared from the spleens of the respective swine, were unsuccessful.

The results of these observations seem to warrant the following conclusions:

1. Although the particular strain of *Br. abortus* utilized for inoculation of the swine has remained consistently pathogenic for guinea pigs, swine appear to possess considerable natural resistance following experimental exposure to this organism.

2. The apparent predilection of the organism for the body of the vertebrae in spontaneous spondylitis is perhaps due to an accidental or casual deposition rather than to an inherent tendency of the organism for elective localization.

3. Although the production of specific agglutinins followed exposure to the infective agent regardless of the method of inoculation, the gradual but definite diminution of the respective agglutinative titres suggests the probability of eventual recovery.

4. The ability of a strain of *Br. abortus* of porcine origin, which was pathogenic for guinea pigs, to induce serious illness and lesions in what might be considered a natural host was not demonstrated.

REFERENCES

[1]Feldman, W. H., and Olson, C., Jr.: Spondylitis of swine associated with bacteria of the Brucella group. Arch. Path., xvi (1933), pp. 195-210.

[2]Feldman, W. H., and Olson, C., Jr.: Isolation of bacteria of the Brucella group from apparently healthy swine. Jour. Inf. Dis., liv (1934), pp. 45-50.

[3]Johnson, H. W., and Huddleson, I. F.: Natural Brucella infection in swine. Jour. A. V. M. A., lxxviii (1931), n. s. 31 (6), pp. 849-862.

[4]Traum, J.: An. Rpt. Chief, B. A. I., U. S. Dept. of Agr. (1914).

[5]Good, E. S., and Smith, W. V.: *Bacillus abortus* (Bang) as an etiological factor in infectious abortion of swine. Jour. Bact., i (1916), pp. 415-422.

[6]Hayes, F.: Some studies in swine abortion. Jour. A. V. M. A., lx (1922), n. s. 13 (4), pp. 435-452.

[7]Howarth, J. A., and Hayes, F. M.: Brucelliasis in the swine herd of the University of California. Jour. A. V. M. A., lxxviii (1931), n. s. 31 (6), pp. 830-848.

[8]Cotton, W. E.: The character and possible significance of the Bang abortion bacillus that attacks swine. Jour. A. V. M. A., lxii (1922), n. s. 15 (2), pp. 179-192.

[9]Weeter, H. M.: Infectious abortion in domestic animals. I. Infection of swine and rabbits. Jour. Inf. Dis., xxxii (1923), pp. 410-416.
[10]Starr, L. E.: Production of agglutinins for *Brucella abortus* in calves, swine and rabbits by skin and mucous membrane contact. Jour. A. V. M. A., lxxxi (1932), n. s. 34 (2), pp. 230-238.
[11]Cotton, W. E., and Buck, J. M.: *Brucella abortus* in the blood stream of swine. No. Amer. Vet., xiii (1932), 2, pp. 35-43.
[12]Huddleson, I. F., Johnson, H. W., and Hamann, E. E.: A study of Brucella infection in swine and employes of packing-houses. Jour. A. V. M. A., lxxxiii (1933), n. s. 36 (1), pp. 16-30.
[13]Makkawejski, W. N., Kardadinowskaja, I. A., and Micheew, N. I.: Ueber den infektiosen abortus (Brucelliasis) des Schweines. Deut. Tierärzt. Wchnschr., xli (1933), pp. 321-327.
[14]James, W. A., and Graham, R.: Porcine osteomyelitis, pyemic arthritis and pyemic bursitis associated with Brucella suis. Jour. A. V. M. A., lxxvii (1930), n. s. 30 (6), pp. 774-782.

Dean Francis Celebrated in Song

Dean Mark Francis, of the School of Veterinary Medicine, A. & M. College of Texas, may truly be said to be "celebrated in song," since Mrs. U. E. Marney, wife of Dr. U. E. Marney, of San Antonio, Texas, wrote and dedicated a "Mark Francis Song," that was sung heartily and enthusiastically by members of the State Veterinary Association of Texas at their Mark Francis Meeting at College Station, June 4-6, 1934.

The words of the song, which is sung to the tune of "When You and I Were Young, Maggie," are as follows:

First Verse

We've gathered today at this grand old shrine,
To honor Mark Francis' Day.
He's achieved a great name in the passing time,
And we sing his praises today.

A companion so true marches by his side
To brighten his way whate'er betide;
Twinkling eyes and a smile that she cannot hide,
The dear one he chose for his bride.

Chorus

Oh! How happy are they in this dear old home,
Where they've spent years that rolled along;
We'll remember the name of Mark Francis long,
And pay tribute singing this song.

Second Verse

Loyal friends gathered 'round from afar and near,
Their hearts filled with happiness,
To pay tribute to him for the work of years,
Accomplished with honor and zest.

The time has been long, and the work well done,
Since Texas claimed him as her son;
Let us join in this program of joy and fun,
While we honor this noble one.

TOXIC PROPERTIES OF GREASEWOOD*

With a Brief Discussion of the Physiological Action of Oxalic Acid and Its Soluble Salts

By VIRGIL A. WILLSON, *Helena, Mont.*
Montana Live Stock Sanitary Board

DESCRIPTION OF THE PLANT

Greasewood, botanically known as *Sarcobatus vermiculatus*, is peculiarly adapted to strong alkali soils and old alkali seeps on which other vegetation is scarce. During the growing season it produces a heavy growth of soft green foliage which has a characteristic offensive odor, especially when the leaves are macerated. In some localities the shrub is reported to grow in excess of nine feet high, while plants from three to five feet high are more common. It does not appear to grow on soils free from alkali and suitable to a better type of vegetation. It is reported in the western states from Washington to Montana and from California to Texas.

The dense growth of green leaves produced during the early spring and summer is observed to fall off soon after the first frost, leaving a skeleton plant of scraggy, spiny branches. Although the plant has been referred to as good forage, it has been our observation that animals will not touch it if a better feed is available. This is true especially in the early spring, before the leaves have grown, and in the late fall and winter, after the leaves have fallen, and the mass of skeleton thorns is neither tempting nor nourishing. It is true, however, that the thorns are largely obscured during the growing season and that an animal may obtain an abundance of the green leaves at this time of year without being materially affected by the thorns.

TYPICAL CASES OF LIVE STOCK LOSSES DUE TO GREASEWOOD POISONING

A loss of sheep from greasewood poisoning was reported to this Department by Dr. N. B. Smith, in June, 1931. Nine hundred sheep owned by Mr. G. B., of Forsyth, were turned out of a shearing-pen onto a pasture of greasewood on Saturday morning. No losses were reported until Sunday noon, when the sheep were allowed to water at a stream of running water. Thirty-five head died within two hours after watering, and 150 more were reported sick at that time. No report was made to this Department of the

*Received for publication, January 12, 1934.

total number of sheep that died. Although the loss of sheep in this case was not so great as has been recorded by other observers, the conditions are similar.

A case is reported in Harper, Oregon, where 1,700 ewes in transit, in October, 1920, were unloaded and allowed to graze from 2 to 5 p. m. on a field barren of all foliage except greasewood. The next morning 1,000 head were dead.[1]

From the reports available it has been observed that sheep which have been kept off feed for some time and are then turned out on a greasewood pasture are the principal sufferers. Sheep and other types of live stock often feed in the same field where there is an abundance of greasewood, but do not touch it, as there is better feed available.

Previous Chemical Work Done

Considerable work concerning the poisonous effects of greasewood leaves on sheep, and including a chemical examination of the leaves for the poisonous principle, is reported in U. S. Department of Agriculture Circular 279, by Marsh, Clawson and Couch.[1] These authors reported the poisonous properties of the plant to be due to the oxalates of sodium and potassium.

Purpose of This Investigation

As no figures were available to indicate the amount of the oxalates present in greasewood leaves, or whether the oxalate content is variable during the season, experiments were started in this laboratory in order to obtain this information.

In June, 1931, a sample of greasewood leaves was taken from a field near Toston and analyzed for oxalic acid and other soluble oxalates. There was no free oxalic acid present, but the oxalates calculated as sodium oxalate amounted to 3 per cent of the undried leaves.

Table I—*Analysis of greasewood leaves.*

Constituent	Sample Taken					
	May 11	May 24	July 5	Aug. 8	Sept. 10	Oct. 15
Moisture in leaves (%)	82.70	84.50	83.70	72.48	73.07	77.50
Oxalates as $Na_2C_2O_4$ on dry basis (%)	10.95	13.15	19.05	17.90	21.70	19.95
Oxalates as $Na_2C_2O_4$ on wet basis (%)	1.89	2.03	3.11	4.93	5.84	4.37
Reaction of moist leaves to litmus	N	N	N	N	N	N

N = Neutral.

In the spring of 1933, it was decided to make several periodic tests of the oxalate content of greasewood leaves from a patch growing on the Masonic Home Farm near Helena, with the idea of determining whether or not the oxalate content remained constant throughout the season and whether or not it was at all times sufficient to cause live stock poisoning. Samples were obtained and analyzed and the results are shown in table I.

A mineral analysis of a composite sample of the leaves taken on different dates (table I) showed the total ash as oxides to be 17.25 per cent of the dried sample.

TABLE II—*Mineral analysis of ash.*

MINERAL	%
Barium as BaO	2.71
Iron and aluminum as Fe_2O_3	2.08
Calcium as CaO	6.02
Magnesium as MgO	4.64
Sodium as Na_2O	67.40
Potassium as K_2O	17.30

DISCUSSION OF RESULTS

The maximum of 21.70 per cent oxalates as sodium oxalate, calculated on the dry basis, of the sample taken in September, is approximately twice as great as the sodium oxalate content of a sample taken in May.

The mineral analysis of the dried leaves shows 17.25 per cent of mineral substance calculated as oxides.

The sodium and potassium oxides constitute 84.7 per cent of the mineral substance of the leaves while the sodium oxide content is approximately four times the potassium oxide content. If the entire amount of sodium and potassium were combined in the plant tissues as the oxalates, they would amount to 30.3 per cent of the dried leaf matter. Since the maximum amount of oxalates as sodium oxalate was 21.70 per cent, it is apparent that a considerable amount of the sodium and potassium are combined other than as oxalates.

The oxalate content of the samples analyzed increases during the growing season and reaches a maximum of 5.84 per cent sodium oxalate in the early part of September. It will be noted that the minimum of 10.95 per cent sodium oxalate on the dry basis made on a sample taken in May is approximately one-half of the maximum sodium oxalate content of a sample taken in

September, while the minimum of 1.89 per cent on the wet basis is less than one-third of the maximum sodium oxalate content on the wet basis. The difference is due to the difference in moisture content at these respective periods. As will be observed, the oxalate content increases during the growing season on the dry basis, but at a lesser ratio than on the wet basis, due to the drying up of the moisture in the leaves during the summer and fall months.

Marsh, Clawson and Couch state that 1.5 pounds of the green plant per 100 pounds of body weight of a sheep will generally cause sickness or death. Assuming an average content of 3 per cent of sodium oxalate in the green leaves, this would represent 20.43 grams, or .72 ounces of sodium oxalate.

The same amount of green leaves containing 1.89 per cent sodium oxalate, which is the smallest amount shown in any analysis, would represent 12.8 grams, or .45 ounces of sodium oxalate.

Although we do not have any figures available to indicate the fatal dose of sodium oxalate for sheep, it is reasonable to suppose that they could be poisoned from a good feeding of greasewood leaves containing 1.89 per cent sodium oxalate. Fatal poisoning of humans has been observed with 2 to 5 grams of oxalic acid and any amount over 15 grams is generally considered fatal if it is retained.[2]

Physiological Action of Oxalic Acid and Its Soluble Salts

The poisonous properties of oxalic acid and its salts are due to the acid oxalate radical (C_2O_4). A brief consideration of three phases of oxalate poisoning will be mentioned here.

1. When taken in large doses, the local action of the poison is one of irritation and corrosion. Death in some cases occurs rapidly and it is believed that the poison in such cases has a violent and fatal effect on the nerve centers, respiratory system and heart action.

2. When taken in either large or small amounts, soluble oxalates are rapidly absorbed by the tissues and taken into the bloodstream. There it combines with the blood calcium, forming an insoluble calcium oxalate. The effect is twofold. First, the calcium is removed from the blood and the tissues and their normal life function is interfered with. The removal of calcium from the blood greatly hinders the normal coagulation period of the blood, and weakens the heart action. Second, a great burden is thrown on the organs of elimination in an effort to remove the excess calcium oxalate precipitated and congestion occurs, especially in the kidneys.

3. When largely diluted and taken in small amounts the corrosive effect of oxalates is negligible or absent and the chronic effect is one of a gradual removal of calcium from the blood and tissues and the subsequent elimination of the insoluble calcium oxalate. This elimination of the calcium oxalate by the urine may be entirely normal in cases where the urine has a distinct acid reaction, but if the urine is neutral or alkaline, as is the case in the herbivora, the accumulation of the insoluble calcium oxalate leads to the formation of urinary calculi of a serious nature.

Although the soluble oxalates are extremely poisonous to the herbivora as well as to the carnivora, and oxalates in the form of oxalic acid and its soluble salts are normal substances in many plants, cases of live stock poisoning by oxalates are not common. This is because of the protecting effect of calcium, which is a necessary constituent of most forms of plant life, and occurs in abundance in many others. The calcium combines with the oxalates to form calcium oxalate which is insoluble in alkalies and organic acids.

Any oxalic acid or other soluble oxalate which combines with calcium in the animal's stomach is changed to a non-toxic insoluble substance, calcium oxalate, and as such is carried through the digestive system without absorption into the blood-stream.

Marsh, Clawson and Couch state that 4.2 pounds of greasewood leaves per 100 pounds of body weight for a sheep was fed for 34 days in connection with other hay without any ill effects, while 1½ pounds per 100 pounds of body weight would ordinarily produce illness or death where fed exclusive of any other feed. The difference is undoubtedly due to the neutralizing effect of the poisonous properties of sodium oxalate by its combination with calcium from the other feed given.

Suggested Treatment

Although treatment in cases of plant poisoning under range conditions is usually quite difficult and severe losses are experienced before one knows either the cause of the loss or the proper treatment, it is suggested that lime water, made either from slacked or unslacked lime, will be of more benefit than any other treatment. The calcium in the lime combines with the oxalates in the stomach, forming the insoluble calcium oxalate which passes through the digestive system without absorption. The excess of the calcium in the stomach is also beneficial in that it is absorbed by the blood to replace the calcium which has been lost by oxalate precipitation.

In this connection it may be added that the feeding of a calcium diet to animals in localities deficient in lime, or feeding on types of feed deficient in calcium, may well be carried out as a preventive as well as a curative measure. In such cases of lime deficiency chronic oxalate poisoning may result from feeds containing much smaller amounts of oxalates than is shown in greasewood. The feeding of bone meal or a similar form of calcium phosphate, as recommended by Dr. W. J. Butler,[3] to animals obtaining a diet deficient in these elements, has a double value in that the calcium and phosphorus are both essential to bone building and that dangers from oxalate poisoning may be avoided by combining such oxalates, if they are present in the stomach, with the calcium from the calcium phosphate.

CONCLUSIONS

1. The toxic effects of greasewood were found to be due to the soluble oxalates of sodium and potassium. The sodium and potassium salts were in the ratio of approximately four to one.
2. The oxalate content of greasewood leaves increases from the beginning of the growing season and reaches a maximum when the plant has matured during the late part of the summer.
3. In all cases where greasewood leaves have been analyzed, the oxalate content is believed sufficient to produce oxalate poisoning if taken internally by sheep in appreciable quantities.
4. Substances of this nature and containing a high percentage of scluble oxalates may be responsible for cases of chronic oxalate poisoning in the form of urinary calculi.

REFERENCES

[1]Marsh, C. D., Clawson, A. B., and Couch, J. F.: Greasewood as a poisonous plant. U. S. Dept. of Agr. Cir. 279.
[2]Webster, R. W.: Legal Medicine and Toxicology.
[3]Butler, W. J.: Live Stock Sanitary Board, Dept. Cir. Letter, Feb. 7, 1934.

Associated Serum Producers Broadcast

The program that is now broadcast daily by the Associated Serum Producers, Inc., South Omaha, Neb., over stations WLS (Chicago), KFAB (Lincoln, Neb.), WHB (Kansas City) and WNAX (Yankton, S. D.), may also be heard over WBNS (Columbus, Ohio). The advertisements, which are now appearing in farm publications throughout the Middle West, will reach more than 2,500,000 farm subscribers—warning them of hog cholera and urging them to coöperate with the veterinary profession in the prevention of this scourge.

A SKETCH OF THE LIFE OF MARK FRANCIS*

By U. E. MARNEY, *San Antonio, Tex.*

History of medicine and medical men is world history. Underlying every great historical event are medical aspects and activities of medical men that are not given proper recognition. Historians tell us that America was discovered through the hope of finding a shorter route to India over which to transport spices. These spices were not for food condiments but were to be used as medicinal agents. The outcome of the colonization of America would have been problematical if smallpox had not ravaged the Aborigines at that time. Cotton Mather, a colonist, said eighty per cent of the Indians died from this disease, and with typical bigotry of the times, thanked God that the affliction had come to the Indians and cleaned them out to make room for better growth.

The foundation of the British Empire and the building of the far-famed English Navy was started through expansion and controversies over the drug trade and territories where the drugs were produced. President A. Lawrence Lowell of Harvard, in summarizing the progress of civilization, said:

> It is hardly an exaggeration to summarize the history of four hundred years by saying that the leading idea of a conquering nation in relation to the conquered was in 1600 to change their religion, in 1700 to change their trade, in 1800 to change their laws, and in 1900 to change their drainage. May we not say that on the prow of the conquering ship, in these four hundred years, stood first the priest, then the merchant, then the lawyer, and finally the physician?

There are pioneers in every field of endeavor—men with vision and foresight, not content to travel the beaten paths—men like Columbus and Daniel Boone; Fulton and Wright; Edison and Marconi, who, ignoring the scoffing of the multitudes at their advanced theories and ideas, and turning away from the smooth surfaced boulevards of ease and convenience, struck off to blaze new trails through underbrush of ignorance, skepticism and opposition.

Mark Francis came to College Station, Texas, in August, 1888, having completed his veterinary course as the first graduate of the Ohio State University, whose Dean was Dr. H. J. Detmers. Postgraduate work also had been taken by Francis at the American Veterinary College, in New York. Before being called upon to organize the Veterinary Department of the Ohio State University, Detmers had been sent to Texas by the U. S. Secretary of

*Presented at the eleventh semi-annual meeting of the State Veterinary Medical Association of Texas, College Station, June 4-6, 1934.

Agriculture to make a short investigation of the fever which later was named tick fever.

When the Board of Control of the Texas A. & M. College decided they needed a man in connection with the newly organized Experiment Station to work on animal diseases, some of them, having met Dr. Detmers while he was in Texas, suggested he be communicated with. Detmers immediately urged Francis to accept the Texas position, which he did. His first work after arriving consisted of some classroom lectures to agricultural students. No provision had been made for investigational work, except that they had hired a man to do it. A room of small dimensions, about 14 by 16 feet, served as office, classroom and laboratory. After the lapse of one year a small adjoining room was provided as a classroom. In this unsuitable place, and with meager equipment, Francis toiled for 15 years.

Early Days in Texas

At this time Texas, except in area, climate and natural resources, was very much unlike the Texas of today. There were no great highway systems and privately owned vehicular conveyances to make it possible to travel the length and breadth of its 266-mile area between the rising and setting of the sun. Electric lights, telephone systems, water service companies and modern conveniences were conspicuous by their absence. We are told that it was not an unusual sight on the main streets of our present major cities for signs to be placed on mud-holes reading "No Bottom"; and there were only three or four graduate veterinarians in the State. This was eight years before x-rays were known and only ten or twelve years after Lister pioneered asepsis in surgery. The discovery of the tubercle bacillus antedated 1888 by only six years and tuberculin was not used until two years later. The first immunization treatment for rabies was given by Pasteur only three years prior to 1888.

Even though Texas had an unlimited supply of natural vegetation over her great area, where cattle would thrive throughout the entire year without additional feeding, the native animals consisted of the nondescript Longhorn, cat-hammed variety, which could not be improved or bred up because of the fever which attacked all animals that were brought into the State. Also, these bovines, such as they were, could not be taken to northern sections due to the fact that a trail of cattle deaths followed the path of their transportation. Francis, with his keen analytical mind and ability to pick the most important feature

of any proposition, readily saw in the cattle fever the thing that must be conquered and relief obtained therefrom if Texas was to prosper as she should as a live stock state.

Attempts at Immunization Against Tick Fever

It is a matter of common information how, year after year, even after the tick was incriminated as the carrier of this fever, Francis worked unceasingly to provide methods both for the eradication of the tick from the Texas range and the immunization of breeding cattle imported to improve the low quality of the native animals. This was a long struggle against much opposition and a great amount of experimentation under adverse conditions. In 1897, after nine years of work, Francis drew blood

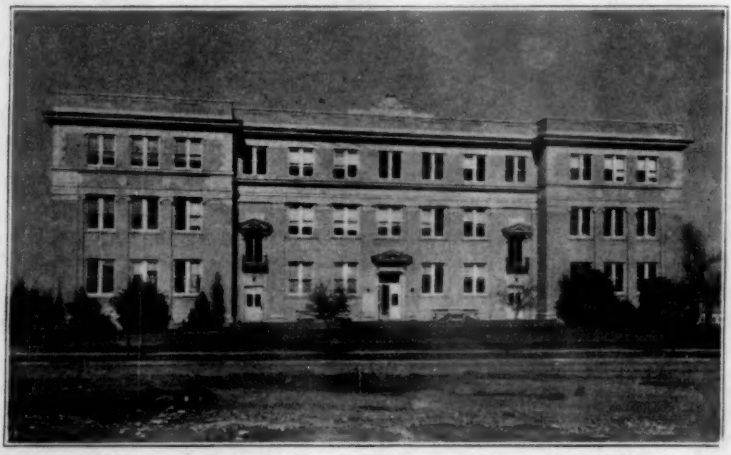

Fig. 1. Francis Hall, Texas A. & M. College, College Station, Texas. Built in 1918.

from a native cow in Brazos County, Texas, and sent it to Dr. U. E. Ward, a practicing veterinarian of Wichita, Kansas, for the injection of ten head of young bulls to be used to head native herds of cows on Texas ranges. When these bulls were shipped to tick-infested territory and all of them, except one, demonstrated a solid immunity and served their purpose well, "Eureka!" was spelled for an important phase of the undertaking in behalf of the cattle industry of Texas.

From then on, large numbers of well-bred animals were similarly immunized for use in ticky territories, with resultant steady and constant improvement in the quality of the cattle of the Southland. Solution of this phase of the cattle-tick problem has

since been duly credited with speeding up the status of the cattle industry twenty years, but it did not mean cessation to Francis; he continued working on what would appear to have been an utter impossibility—the elimination of the piroplasma-carrying parasite. It is a matter of record how different agents and methods were tried in efforts to find a suitable parasiticide to kill the ticks on the animals. Even electricity was used on some of these experiments.

Francis was prevented from using arsenical solution for this purpose; later this proved to be the preparation *par excellence,* not only for freeing southern cattle of ticks so that they would be acceptable in other states, but also for cleaning up vast areas, zone by zone, with the inevitable result that the tick will be entirely eliminated from the nation. The writer personally knows of one commercial house which at one time had in its possession $2,500,000 worth of county warrants from Texas counties which had been rendered in payment for arsenical dip used in dipping cattle in dipping-vats; said vats also having been first introduced by Mark Francis in dipping cattle for tick eradication.

Growth of the Cattle Industry in Texas

It is unusual for the ideas and innovations of great pioneers to be adopted during their lifetime, but we have an exception in the case of Mark Francis. He has seen the cattle industry of his state grow, from what has already been described, to a beef cattle valuation of $160,500,000, and the dairy cattle to over $55,000,000. For years, beef cattle and dairy products have gone from Texas in a veritable stream to feed millions of the population of the world both in peace and in war.

He has seen baby beef exhibited and first prizes taken in our great live stock shows by Four-H Club members from counties where the ribald population and flagrant press strenuously opposed tick eradication which made all of this progress possible.

Only the attainments of Mark Francis with reference to the tick fever problem have been mentioned. Even though this has been an accomplishment of major magnitude, the work of Francis has by no means been limited to it alone. We are now assembled at one of the greatest agricultural colleges in the world and every step of its growth through the years since 1888 has been made possible in no small degree by the subject of this sketch. In 1916, the present School of Veterinary Medicine was established, of which Mark Francis was made Dean. In 1918, Francis was asked if he could use an appropriation for building, and what amount.

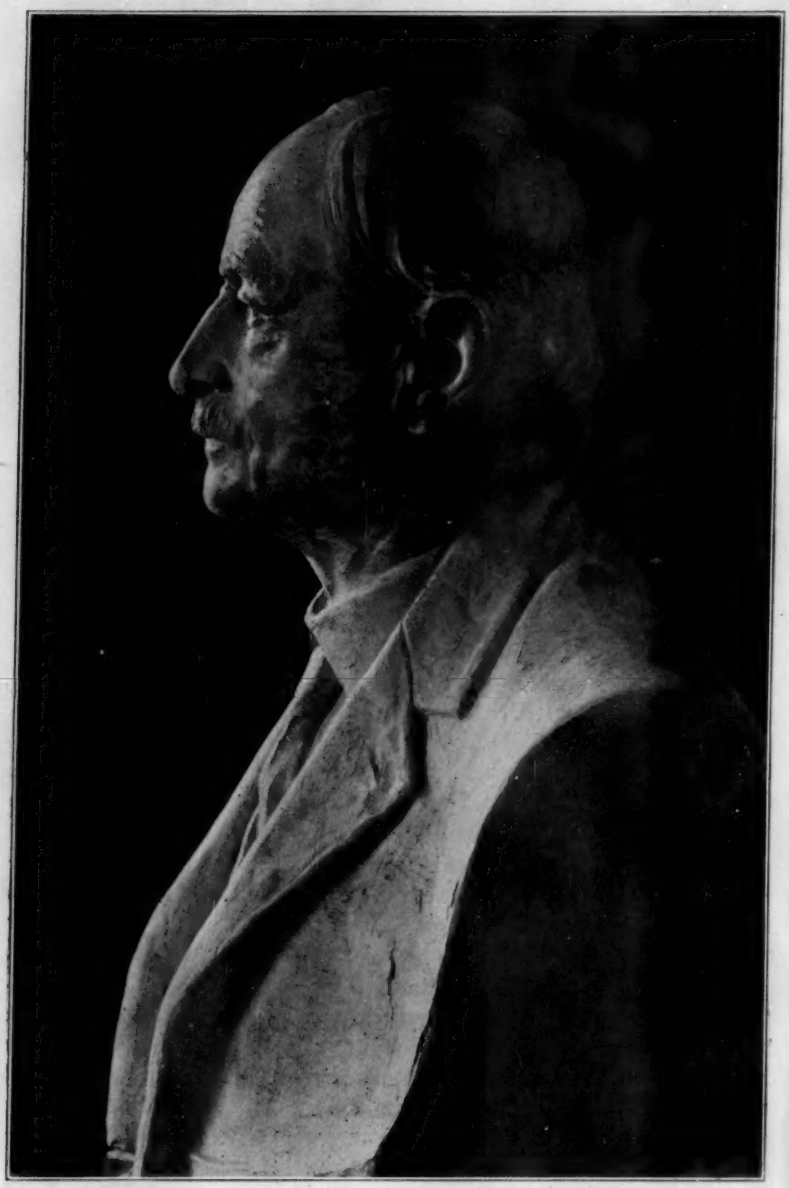

FIG. 2. Bust of Dr. Mark Francis, placed in the Animal Industries Building, Texas A. & M. College, 1933.

The answer was in the affirmative and the amount he specified was $25,000 or $30,000. Almost $100,000 was given and Francis Hall was the result. In 1932, a building program was instituted and he again was asked for an estimate on desired buildings and $100,000 was his answer. T. O. Walton, president of A. & M. College, told the Board of Control that the name of Mark Francis was worth twice this amount. The result has been the erection of the Veterinary Hospital group of buildings, costing $180,000, and the Dissection Laboratory, costing $20,000, bringing the buildings and equipment of the School of Veterinary Medicine to first or second rank among such institutions in the world. The fact that a bust of Mark Francis has been placed in the new $250,000 Animal Industries Building speaks its own significance.

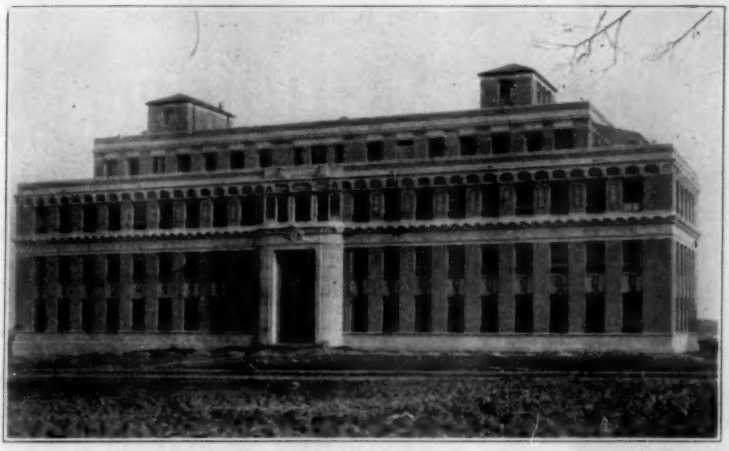

FIG. 3. Animal Industries Building, Texas A. & M. College, College Station, Texas.

As Hill, the railroad king, visualized the great Northwest; Edison, cities, towns and homes lighted with electricity; Carnegie, Frick, Cory and Schwab, the future of the steel industry, and Ford, a motorized civilization—so Mark Francis will go down in history as the man who dreamed of, and did the major portion of making possible, the great cattle industry of the South.

William O. Thompson, president of Ohio State University from 1899 to 1925, made the statement that if the Ohio State University, during all the years of its existence, had done nothing more than give Mark Francis to the world, her mission had been worth while.

President T. O. Walton, of this College, stated a few years ago that of all the professors and workers in connection with the Texas A. & M. College, Mark Francis was considered the most intellectual and was the most loved by his fellowman.

This sketch of the life of Mark Francis has, of necessity, been comparatively brief. A versatile writer could easily produce a book of potent and informative data on his activities and attainments, and this short treatise should not be understood and is not intended as an obituary. Only a few months ago, we stood with Mark Francis on this campus and as he viewed the college

FIG. 4. Main Building, School of Veterinary Medicine, Texas A. & M. College, College Station, Texas. Built in 1932.

buildings and dormitories, he said: "Doctor, I love it here at College Station." And in discussing the buildings and equipment of the School of Veterinary Medicine as they now stand, he has repeatedly said: "We have got started now. We are ready to do something."

Mr. President, it has been said that a great man is not without honor except in his own country. Let us not be the last to extend honor even though he is one of us—our Mark Francis. I beg to make the motion, that the regular order of business of this meeting be suspended and Mark Francis be elected to honorary membership in the State Veterinary Medical Association of Texas.

12th International Veterinary Congress
New York—August 13-18, 1934

EQUINE ENCEPHALOMYELITIS CROSS-IMMUNITY IN HORSES BETWEEN WESTERN AND EASTERN STRAINS OF VIRUS*

By EDWARD RECORDS *and* LYMAN R. VAWTER
Nevada Agricultural Experiment Station
Reno, Nevada

Recent reports by Ten Broeck and Merrill[1] and Giltner and Shahan[2] indicate that the type of equine encephalomyelitis virus encountered in the Atlantic states in 1933 was immunologically dissimilar to the virus encountered in the western states by Meyer, Haring and Howitt[3] and Records and Vawter.[4-6] Preliminary work of our own on guinea pigs also indicated that this was the case. While there is no reason to question these results, as obtained with small, laboratory animals, to our knowledge no one has so far reported cross-immunity experiments on horses.

Inasmuch as we had eight horses available which were known to be more or less immune to the western type of encephalomyelitis, it appeared opportune to test the resistance of these horses to the eastern type of virus.

HISTORY AND PREPARATION OF VIRUS USED

Through the courtesy of Dr. H. W. Schoening, of the Pathological Division, U. S. Bureau of Animal Industry, we had available two strains of the eastern virus of known history.

The Delaware strain (D-I) and the Maryland strain (Md-I) were both recovered in September, 1933. The Delaware virus was received in the form of brain tissue from the third guinea-pig passage; it had not been passed through horses. The Maryland strain had at least six guinea-pig passages but no horse passages when received.

These two strains of virus reached us in November, 1933, and have since been maintained by monthly passages through guinea pigs. The Delaware virus is consistently the more virulent of the two for guinea pigs, according to our observations, causing paralysis, prostration and death in 48 to 60 hours following intracranial injection of 0.2 cc of a 2 per cent brain suspension. Furthermore, the Delaware strain readily infects guinea pigs by skin scarification, intraperitoneal and intranasal administration, although only a small percentage of infections occur following subcutaneous or intradermal injection.

*Received for publication, June 11, 1934.

The Delaware strain, therefore, was selected for this work. The lot of virus used on our experiment horses in May, 1934, was from the ninth guinea-pig passage.

In order to insure a uniform virus suspension at all times during this experiment, a 20 per cent brain virus suspension was prepared by pooling the brains of four guinea pigs which had been killed during the paralytic stage following either intracranial or intranasal injection with Delaware virus. The brain tissue was finely ground in a mortar with aseptic precautions and finally suspended in sterile 50 per cent phosphate glycerin, pH 7.4. This stock virus suspension was stored at 5° C. It proved sterile on cultural tests.

We have already reported that similarly prepared suspensions of western virus remained active for at least four months when stored at 5° C.[5]

A preliminary test of this glycerinated stock virus suspension, although not carried to an m.l.d. determination, showed 0.2 cc of a 1:1,000 dilution to be infective for 400-gram guinea pigs upon intracranial injection. Titration of the glycerinated stock virus at the end of the experiment revealed an m.l.d. of 0.2 cc of a 1:100,000 dilution for 400-gram guinea pigs upon intracranial injection.

A 1 per cent brain virus suspension was prepared from the finely ground glycerinated stock virus by dilution with phosphate saline pH 7.4. This dilute saline suspension was allowed to extract for twelve hours at 5° C., followed by centrifugation for one-half hour at high speed. The supernatant fluid was used for inoculation purposes, a portion being filtered through two Berkefeld N. filters. The filtrates were pooled and proved sterile on cultural test.

History of Horses Used in Experiment

Some of the western virus-immune horses used in this cross-immunity experiment were originally used in the experimental study of equine encephalomyelitis immunization recently reported by us.[6] They were all range-bred horses with part Thoroughbred ancestry, ranging in age from two to twelve years, averaging about 950 pounds in weight, and were in excellent physical condition.

Horse 8352 received, on May 5, 1933, a single subcutaneous injection of 10 cc of a 2 per cent brain-virus suspension of our virus A, mentioned in a recent publication.[6] This horse survived two 10-cc intranasal instillations of 2 per cent virus B[6] suspension on March 9, 1934.

Horses 8355 and 8356, receiving the same subcutaneous virus injection as 8352 on May 5, 1933, survived without symptoms two 10-cc intranasal instillations of a 2 per cent suspension of virus B, on March 9, 1934, and an intracranial injection of 5 cc of a 2 per cent suspension of virus B on April 13, 1934.

Horse 8359 received 5 cc of a 2 per cent suspension of virus A subcutaneously on May 5, 1933, and two weeks later 10 cc of a 2 per cent suspension of virus B subcutaneously. This individual survived an intranasal instillation of 50 cc of a 2 per cent suspension of virus A in August, 1933, and a 5-cc intracranial injection of a 2 per cent suspension of virus B in November, 1933. The serum of this individual neutralized western virus *in vitro*.

Horses 8362 and 8364 received the same subcutaneous injections of virus on the same dates as 8359 and survived without symptoms the same intranasal and intracranial administration of virus on the same dates as 8355 and 8356.

Horse 8367 received the same subcutaneous virus injections on the same dates as 8359, 8362 and 8364 and survived two intranasal instillations of 10 cc of a 2 per cent suspension of virus B on March 9, 1934, without symptoms.

Horse 8543 received a single subcutaneous injection of 20 cc of a 2 per cent suspension of virus B on July 20, 1933, and survived an intracranial injection of 5 cc of a 2 per cent suspension of the same virus on August 11, 1933, without symptoms. The serum of this horse neutralized western virus *in vitro*.

Non-immune control horses in the same group, given western virus intranasally and intracranially during immunity tests in March and April, 1934, developed typical virus syndromes and were destroyed when moribund. It is apparent that all of the above horses possessed a high though perhaps variable degree of immunity to the western virus.

It is considered impossible that any of these horses, immune to western virus, could have had any natural exposure to eastern virus prior to the cross-immunity test. We limited our preliminary animal passages of eastern virus entirely to guinea pigs, which were confined indoors about one mile from the experiment farm where the horses were quartered.

Horse Immunity Tests

Table I shows the details pertaining to the exposure of these horses to the eastern (Delaware) virus. The control horses used were brought in, immediately before the start of the cross-immunity experiment, from an isolated range district in northern Nevada where equine encephalomyelitis has not been known to

occur. They were mature unbroken geldings in excellent condition ranging in age from three to ten years.

The virus filtrate was administered intranasally to two immune horses and intracranially to another immune horse under anesthesia.

The unfiltered 1 per cent virus suspension was administered intracranially under anesthesia to three immune horses and one

TABLE I—*Cross-immunity tests on horses.*

HORSE	TESTED	INOCULUM	METHOD	RESULTS
8359 (Immune)	5–10–34 10 a.m. and 3 p.m.	20 cc virus filtrate	Intranasal	Survived without symptoms
8362 (Immune)	5–10–34 10 a.m. and 3 p.m.	20 cc virus filtrate	Intranasal	Survived without symptoms
8355 (Immune)	5–10–34 10 a.m. and 3 p.m.	20 cc virus suspension (1%)	Intranasal	Survived without symptoms
8543 (Immune)	5–10–34 10 a.m. and 3 p.m.	20 cc virus suspension (1%)	Intranasal	6th day: T 106, slightly dull 7th day: T 102, appetite normal No further symptoms. Full recovery
8356 (Immune)	5–12–34	20 cc virus filtrate	Intracranial	6th day: T 104, no other symptoms 7th day: T 100, no further symptoms. Full recovery
8364 (Immune)	5–12–34	5 cc virus suspension (1%)	Intracranial	2nd to 5th day: T 105, dull but no motor disturbance 7th day: T 101, no further symptoms. Full recovery
8352 (Immune)	5–23–34	5 cc virus suspension (1%)	Intracranial	1st day: T 105, dull but still eating 2nd day: T 104, increased dullness. Tremor of triceps muscles 4th day: locomotor ataxia appeared 5th day: moribund. Destroyed
8367 (Immune)	5–23–34	5 cc virus suspension (1%)	Intracranial	1st day: T 105, dull 2nd day: T 105, muscle tremors, lip palsy 3rd day: prostrate and moribund. Destroyed
9661 (Control)	5–23–34	5 cc virus suspension (1%)	Intracranial	1st day: T 103.4, very dull but still eating 2nd day: T 104.6, very dull, tremor of triceps muscles, lip palsy 3rd day: prostrate and moribund. Destroyed

TABLE I—*Cross-immunity tests on horses—Concluded.*

HORSE	TESTED	INOCULUM	METHOD	RESULTS
9662 (Control)	5–23–34 10 a.m. and 3 p.m.	10 cc virus suspension (1%)	Intranasal	2nd day, T 104, no other symptoms 3rd day: T 103, triceps tremor 5th day: T 106, very dull, increased tremor, lip palsy, anorexia 6th day: T 101, braced against fence at 6 a.m. Sudden collapse with respiratory and cardiac distress, moribund at 11 a.m. Destroyed
9663 (Control)	5–23–34 10 a.m. and 3 p.m.	10 cc virus suspension (1%)	Intranasal	6th day: T 101, muscle tremors 7th day: T 104.4, very dull, extensive muscle tremors, slight lip palsy, anorexia 8th day: T 101.6, ataxic symptoms, recumbent part of the time 9th day: T 101, down but could rise, ataxic movements, impaired vision 10th day: T 101, more animated but still dull, ate and drank sparingly 16th day: ataxia and impaired vision still present, probable permanent disability

non-immune horse and by intranasal instillation to two immune horses and two non-immune controls.

Concurrent with each horse inoculation, control guinea pigs were injected intracranially with 0.2 cc of the unfiltered 1 per cent virus suspension and the Berkefeld filtrate. All of the control guinea pigs developed the characteristic virus syndrome and died.

DISCUSSION OF RESULTS

From the foregoing experiment it is apparent that six of the eight horses immune to the western type of encephalomyelitis virus were also resistant to the eastern type of virus. All of the non-immune controls developed characteristic symptoms; two were destroyed and one remains disabled. Although three of the six surviving immune horses showed very mild transitory symptoms, there was no disturbance of motility or serious impairment

of appetite. All of them returned to normal by the eighth day and recovered without disability.

The two horses, which were presumably immune to western virus but which became prostrate following the intracranial injection of eastern virus, had not had any reinforcement by virus exposure other than intranasal exposure to virus in March, 1934, about two months prior to the eastern virus exposure. To what extent intranasal or intracranial administration of virus may reinforce the resistance is not known. We have observed that guinea pigs, surviving the intracranial or intranasal administration of sublethal amounts of virus, are not uniformly immune to subsequent severe virus exposure.

The control horse, which received Delaware virus intracranially, was moribund 72 hours after injection. This was about 48 hours earlier than is usual following intracranial injection with western virus.

The two control horses which received Delaware virus intranasally developed typical symptoms on the fifth and seventh days, respectively, which is a slightly shorter incubation period than occurs in horses as a result of intranasal exposure to western virus. Otherwise, there were no essential differences in the symptomatology or gross anatomical changes compared to western virus cases.

To date we have successfully induced one non-fatal and ten fatal infections in horses by the intranasal instillation of western virus, using two different strains. In two instances, a single 5-cc instillation of a 1 per cent virus suspension resulted in fatal infection.

Apparently from our limited experience with eastern virus, infection can quite as readily occur following its intranasal instillation as shown by control horses 9662 and 9663. The fact that the eastern type of virus as well as the western type will cause infection in the horse by the intranasal route leads us to conclude that the olfactory tract is one of the probable routes of entry of the virus under natural conditions.

CONCLUSIONS

1. Six out of eight horses, immune to western types of equine encephalomyelitis virus, also proved immune or resistant to the eastern type of virus.

2. Infection with the Delaware strain of eastern virus was readily induced in three non-immune control horses, either by intracranial injection or intranasal instillation of a 1 per cent virus suspension. Two of the controls became prostrate and were

destroyed when moribund. The other control which received virus intranasally developed grave symptoms followed by incomplete recovery. Ataxia still persists, indicating a probable permanent disability.

3. The disease resulting from the administration of the eastern virus ran a more rapid course than that produced by the western type. Otherwise, there are no essential differences in the clinical syndrome produced by the two viruses.

4. It appears from cross-immunity observations made on horses, the natural hosts for these viruses, that any difference between the eastern and western types of virus may be only one of virulence rather than specific immunologic dissimilarity.

REFERENCES

[1]Ten Broeck, C., and Merrill, M. H.: A serological difference between eastern and western equine encephalomyelitis virus. Proc. Soc. Exp. Biol. & Med., xxxi (1933), 2, pp. 217-220.

[2]Giltner, L. T., and Shahan, M. S.: The immunological relationship of eastern and western strains of equine encephalomyelitis virus. Sci., lxxviii (1933), 2034, pp. 587-588.

[3]Meyer, K. F., Haring, C. M., and Howitt, B.: The etiology of epizoötic encephalomyelitis of horses in the San Joaquin Valley in 1930. Sci., lxxiv (1931), 1913, pp. 227-228.

[4]Records, E., and Vawter, L. R.: Equine encephalomyelitis anti-serum. Jour. A. V. M. A., lxxxii (1933), n. s. 35 (4), pp. 608-616.

[5]Records, E., and Vawter, L. R.: Equine encephalomyelitis. Nev. Agr. Exp. Sta. Bul. 132 (1933).

[6]Records, E., and Vawter, L. R.: Equine encephalomyelitis immunization. Jour. A. V. M. A., lxxxiv (1934), n. s. 37 (5), pp. 784-798.

Swindler Reported in Indiana

Veterinarians are warned to be on their guard against an imposter representing himself as M. W. Hoffman, and an agent for a company of that name, said to be located at 1026 Arch Street, Philadelphia, Pa. It is reported that letters mailed to this address have been returned to the writers unclaimed.

The said Hoffman travels around, calling on veterinarians, and tries to sell them instruments, microscopes, etc., said to be stock disposed of by the Army following the war. He agrees to sell almost anything and will take used articles as part payment. He then asks for a down payment in cash of about 30 per cent, agreeing to ship the purchased goods at once and to give a 5 per cent discount on the balance due if paid in ten days. This imposter is described as being about five feet, five inches tall, weighs about 135 pounds, has a good line of conversation and claims to have been connected with the Intelligence Department of the Army during the war.

Take no chances with anybody answering the above description. When last reported, this swindler was operating in Indiana.

THE ETIOLOGY OF FOWL PARALYSIS (NEUROLYMPHOMATOSIS GALLINARUM-PAPPENHEIMER), LEUCOSIS AND ALLIED CONDITIONS IN THE DOMESTIC FOWL*

Preliminary Report

By M. W. EMMEL, *Gainesville, Florida*
Agricultural Experiment Station

Since fowl paralysis and leucosis were first observed, they have become progressively more prevalent and at the present time they constitute a major disease problem in almost all parts of the world. Their etiology has remained unknown. It has been only in recent years that there has been doubt that they are the separate and distinct disease entities which they have long been regarded.

Ellermann[1] was the first investigator to show that leucosis could be transmitted with a certain degree of success by means of emulsions and cell-free extracts of affected tissues. This observation has been confirmed by many investigators. Likewise numerous investigators also agree that fowl paralysis may be transmitted in a similar manner. Patterson, Wilcke, Murray and Henderson,[2] however, were the first to show that both diseases may be transmitted by the same filtrable agent. Johnson[3] has recently confirmed this observation.

In a series of papers, the first of which are now being prepared for publication, the author expects to submit evidence to show that: (1) the same etiological agent is responsible for fowl paralysis, leucosis in its various forms, and other allied conditions in the fowl; (2) the primary etiological agent is non-specific in that any one of a number of microörganisms of the paratyphoid and typhoid groups of bacteria may serve as an inciting agent; (3) enteritis, most commonly caused by intestinal parasites, is a necessary predisposing factor; (4) cases of fowl paralysis, leucosis, and allied conditions so induced are comparable to naturally occurring cases.

Microörganisms of the paratyphoid and typhoid groups may be isolated from the intestinal tract of the fowl, particularly young birds, in some cases of enteritis and in extremely acute cases of fowl paralysis. It is the contention of the author that the inflammation of the intestines accompanying intestinal parasites provides an avenue of infection for the primary etiological agent.

*Received for publication, June 14, 1934.

This agent passing directly into the blood-stream induces hemocytoblastosis (in the case of leucosis). When hemocytoblastosis is once established it is sustained by the continued entrance of the primary etiological agent into the blood-stream. When the proper momentum has been developed, the disease progresses under its own impetus, with the result that any of the various forms of leucosis may become manifest. Erythroleucosis represents the most severe degree of infection. The exact method by which the etiological agent induces fowl paralysis is not clearly understood at this time. However, this disease represents the lightest degree of infection.

The type of disease induced, *i. e.*, fowl paralysis, erythroleucosis, myeloid leucosis, and allied conditions, depends upon: (1) the resistance of the individual bird; (2) the severity and duration of the enteritis; (3) the inherent properties of the particular strain of paratyphoid or typhoid organism acting as the primary etiological agent; and (4) the number of organisms, rate of entrance, and the period over which such organisms gain access to the blood-stream of the bird.

While it is considered important that certain members of the paratyphoid and typhoid groups of bacteria are the primary etiological agents for fowl paralysis and leucosis, it is of equal importance that this research opens an entirely new field for the epidemiologist who is interested in the relation of these organisms to human health. Furthermore, the author believes that this investigation offers a plausible explanation of the etiology of leukemia in the human.

REFERENCES

[1]Ellermann, V.: The leucosis of fowls and leukemic problems. (London, Gyldendal. 1921.)

[2]Patterson, F. D., Wilcke, H. L., Murray, C., and Henderson, E. W.: So-called range paralysis of the chicken. Jour. A. V. M. A., lxxxi (1932), n. s. 34 (6), pp. 747-767.

[3]Johnson, E. P.: The etiology and histogenesis of leucosis and lymphomatosis of fowls. Va. Agr. Exp. Sta. Tech. Bul. 56 (1934).

Parrot Buried with Mistress

Polly, the pet parrot of Mrs. Bertha Robinson for 50 years, was not left behind when his mistress died in Cincinnati, Ohio, recently, but was chloroformed and buried in a satin-lined casket at the foot of her grave. The bird, which spoke both English and German, had been given to Mrs. Robinson by her husband on their wedding day.

SPONTANEOUS GOUT IN TURKEYS*

By CARL F. SCHLOTTHAUER *and* JESSE L. BOLLMAN
Division of Experimental Medicine
The Mayo Foundation, Rochester, Minnesota

Both visceral and articular gout have been observed in birds, the former having been the more prevalent. Hutyra and Marek[1] stated that gout occurred frequently in birds, and that it had been observed in reptiles and alligators, in a few old dogs, and in a horse. Among domesticated fowl, they stated, it occurred frequently in chickens, less commonly in water fowl; it rarely was seen in pigeons. They stated also that gout frequently has been observed in the birds of prey in zoölogical gardens. On the other hand, it rarely has been seen in birds kept in the house, and it never has occurred in wild birds living at large. Kaupp[2,3] stated that gout was common in birds that were force-fed or given a diet rich in protein and in old birds in which there was more or less impairment of the eliminating action of the kidneys. He stated also that gout occurred more often in flesh-eating than in fish-eating birds. He mentioned having observed gout in a group of five-year-old capons that had run at large and had received a grain diet. Much of the grain had sprouted. Ward and Gallagher[4] agreed with the foregoing authors on the occurrence of gout in chickens, water fowl, pigeons and in caged, wild birds. They mentioned protein diets, rich and unbalanced diets, close confinement, and hereditary predisposition, as etiologic factors. They referred to the observations of Hebrant and Antoine, who noted articular gout in a two-year-old cock which had been receiving a diet of corn and wheat, a protein-poor diet; they also referred to Kionka who had produced gout in chickens experimentally with a diet of horse flesh.

During April and May of 1933, we observed spontaneous gout in several groups of turkeys. Typical tophi and articular involvement were noted. The uric acid content of the blood was deter-

*Received for publication, January 20, 1934.

mined in all affected, and in some apparently normal turkeys; the uric acid content of the material in the tophi, eggs, urine and in the feces was determined in some.

The following is a brief description of the flocks of turkeys in which we observed gout:

Flock 1: Five hens two years of age and a tom one year of age. Gout developed only in the male.

Flock 2: Thirty hens and eight toms. Gout developed in four hens and in four toms of this group. All of these birds were hatched from eggs produced by the hens of flock 1 during the spring of 1932 and they were one year of age.

Flock 3: Two hundred eighty hens and thirty toms. These birds were all one year of age. Gout developed in a large number of these birds, approximately 5 per cent, and the sexes were affected in about equal proportions.

Flocks 4 and 5: One hundred twenty hens and twenty toms, all one year of age. Some of these birds were hatched from eggs obtained from flock 1, but none of these birds was observed to have gout.

All five flocks were confined to outdoor lots which were large enough to permit rather free exercise. After March 1, all flocks received the same commercial laying mash, containing 20 per cent protein.

The first symptoms noted in the affected turkeys were lameness and difficulty in arising from the ground. In the early stages the condition appeared to be so painful that the turkeys spent most of the day sitting on the ground. The attacks of pain seemed to be intermittent, as the turkeys would move about normally for a few days and then resume their characteristic position for two days to a week. Within a few days after the appearance of the first symptoms, small tophi were observed, usually on the feet. These tophi gradually enlarged, but the enlargement in itself seemed to bear no relationship to the symptoms observed. Symptoms were observed also in turkeys in which there was no change in the size of the tophi. We observed no diminution in the size of the tophi in any turkey, but it is possible that some did diminish in size.

The tophi (figs. 1, 2 and 3) and joint cavities contained masses of white, semi-solid or paste-like material not unlike the urate deposits commonly noted in the feces of birds. In some of the turkeys that were affected badly, this same material was deposited subcutaneously, and over the fascia of the muscles of the thighs and wings it was deposited in a layer 0.2 to 0.4 mm in thickness. Articular involvement was noted usually in the feet and in the

legs, although in some the articulations of the wings also were involved. Visceral gout was not observed, but it is possible that some turkeys in these flocks did have visceral gout which escaped observation, since a marked increase in the uric acid content of the blood was noted in some birds that otherwise appeared to be normal. The uric acid content of the tophi was found to be from 4 to 5 per cent of the moist weight of the pasty material easily scraped from the tophi or joint cavities. From 20 to 30 per cent of the dried solid material removed was found to be uric acid.

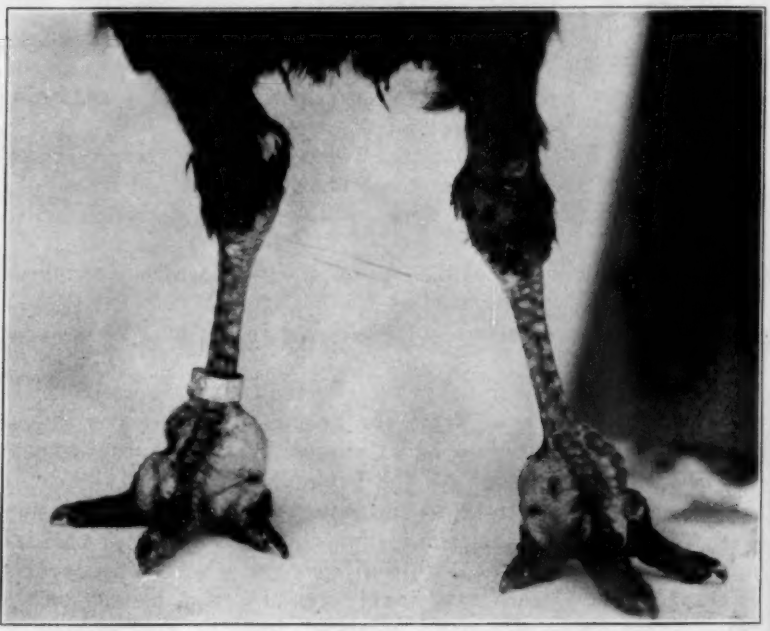

FIG. 1. Involvement of both feet with gouty lesions of about six weeks duration.

Histologic examination of the tophi revealed that they were composed of numerous sheaves of needle-like crystals enclosed in connective tissue of varying density.

All of the turkeys that were observed to have definite tophi were found to have increased amounts of uric acid in the blood. Normal turkeys from the flocks in which no gout was observed were found to have less than 2.0 mg of uric acid per 100 cc of blood. Some turkeys with tophi were found to have as much as 16.0 mg of uric acid per 100 cc of blood, whereas others with as many tophi had as little as 3.0 mg of uric acid per 100 cc. Sev-

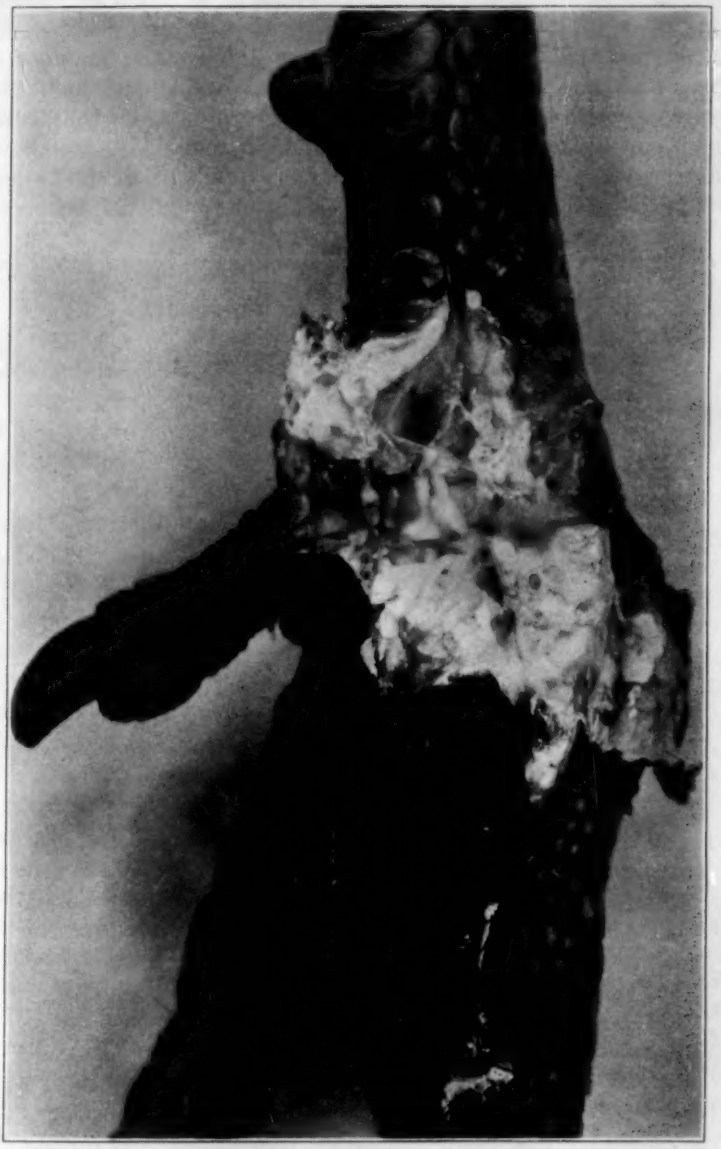

FIG. 2. The foot of a gouty bird with the skin reflected to show subcutaneous deposits of white, pasty material.

eral turkeys were observed in which the uric acid content of the blood rose in the course of several weeks to as much as 16.0 mg per 100 cc and later decreased to about 4.0 mg. During the period of increasing uric acid content of the blood, tophi were increased in size and number, but no decrease in the tophi was observed with decreasing uric acid content of the blood. Symptoms also were present during both periods, although the impression was gained that the symptoms were less frequent during the period of decreasing uric acid content of the blood. We were unable to determine the cause of the change in the amount of uric acid as the turkeys remained under identical conditions during the entire course. The excretion of uric acid varied

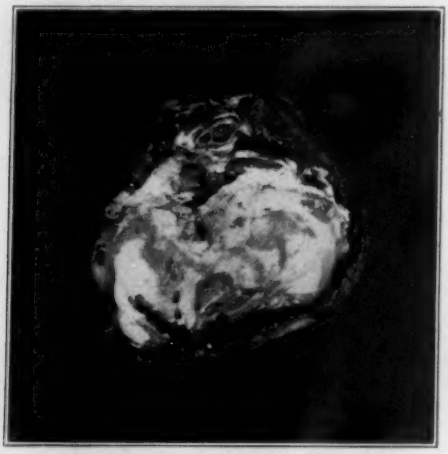

FIG. 3. Gouty deposits in the articulation of the knee.

from 0.6 gm per bird per day to 2.8 gm. Most of this variation appeared to follow similar variation in the amount of food taken and seemed to bear little relation to the amount of uric acid in the blood. The uric acid in the blood in some cases increased; in others, it decreased, and in others it was unaltered during periods of widely divergent excretion of uric acid.

We observed several normal-appearing turkeys in which the uric acid content of the blood was found to be as high as 9.0 mg per 100 cc. Continued observation of these turkeys revealed that they all developed tophi, although several of them maintained high levels of uric acid content in the blood for as long as eight to ten weeks before any evidence of gout became visible. Several

normal-appearing turkeys with increased uric acid content of the blood were confined to cages. High levels of uric acid content in the blood and tophi developed in all of these birds. In most other turkeys which were allowed to remain in their pens gout did not develop so rapidly, although the development of gout in some of these turkeys was almost as rapid as in those confined to cages.

The fertility of the eggs from the flocks with gout was greatly diminished, and there was a high death-rate among the poults which were hatched from these eggs. The infertility may be explained, at least in part, by the inactivity of the males, owing to the presence of symptoms of gout. Eggs obtained from hens with increased uric acid content of the blood had increased amounts of uric acid in both the whites and the yolks. Although the uric acid content of the eggs did not approach the uric acid content of the maternal blood, it was found in amounts as high as 1.0 mg per 100 gm of egg, which is much higher than that found in eggs of the same age from normal turkeys. It is probable that the infertility of these eggs was a result of associated conditions rather than the direct influence of increased uric acid content.

The irregular occurrence of gout in these flocks of turkeys is not easily explained. One might suspect some hereditary influence, since many of the turkeys were descendants of one small flock. Opposed to this idea, however, is the fact that gout failed to develop in many other turkeys of the same origin. Similar criticism of other factors that might be considered as etiologic is also apparent. Diet, age, degree of infection, and exercise undoubtedly play a definite part in the incidence of gout, but in these flocks the conditions were similar as far as could be determined. There appeared to be no predilection as to sex or age in the affected birds. In the hope of elucidating some of these factors, we are attempting to produce gout in turkeys under better controlled experimental conditions.

References

[1]Hutyra, F., and Marek, J.: Special Pathology and Therapeutics of the Diseases of Domestic Animals. (2nd ed., Alexander Eger, Chicago, 1920), iii, pp. 196-197.

[2]Kaupp, B. F.: Poultry Diseases. (4th ed., Alexander Eger, Chicago, 1927), pp. 393.

[3]Kaupp, B. F.: Poultry Diseases. (5th ed., Alexander Eger, Chicago, 1929), pp. 251-253.

[4]Ward, A. R., and Gallagher, B. A.: Diseases of Domesticated Birds. (The Macmillan Co., New York, 1926), pp. 150-152.

STEER'S STOMACH STORES SHOTGUN SHELLS*

By L. F. CONTI, *Los Angeles, Calif.*
Los Angeles County Live Stock Department

Since launching its campaign against avoidable deaths from so-called "hardware disease," the Los Angeles County Live Stock Department has received many interesting specimens, taken from the digestive apparatus of dairy cattle which were killed by for-

Fig. 1. Material found at slaughter in the stomach of a steer.

eign bodies. The annual dairy cattle losses from ingestion of pieces of baling wire, nails, screws, copper tag fasteners, etc., are enormous. The department has endeavored to acquaint dairymen with the proper manner of opening bales of hay, to prevent nails

*Received for publication, March 5, 1934.

from getting into feed mangers during barn construction or repair, and the picking up of fence-wire scraps after repairs, etc.

The accompanying photograph (fig. 1) shows material obtained from the stomach of a range steer from Utah, recently slaughtered in a Los Angeles packing-house. The condition is somewhat different from that found in our ordinary "hardware disease" of dairy cattle, in that it pictures a clear-cut case of pica, or depraved appetite, due in all probability to an insufficient supply of nutrient salts in the ration. In cases of pica, the animal searches for foreign objects to satisfy a craving for the deficient nutrients, while in "hardware disease" cattle in most cases unintentionally swallow metallic objects which, through carelssness, are permitted to get into the feed.

The material collected from this steer consisted of 14 brass shotgun shell bases, two rifle cartridges, three shotgun shell caps, nine pieces of iron and brass, a belt buckle, seven pieces of stone and seven pieces of bone. No ill effect upon health had been noted in this case nor were there any lesions of the digestive system or other organs as a result of the presence of the mass of foreign material.

Sherlock Holmes would say, "There's good hunting where that steer came from."

PAPILLOMATOSIS IN A BOVINE*

By HARRY MORRIS,† J. A. BAKER *and* J. A. MARTIN
Department of Animal Pathology, Louisiana State University
Baton Rouge, La.

On August 10, 1933, a two-year old Hereford bull was received at the laboratories of the Animal Pathology Department of the Louisiana State University. This animal was affected with what is commonly known as warts. The bull was sent in from the extreme southern portion of this State, where a low marshy condition exists.

Aside from the multiple papillomatous growths which completely covered the lower portions of all four limbs (as shown in figure 1), the animal appeared to be in fairly good physical condition. Small warty growths were present also on the scrotum.

The animal was kept under observation in a dry, well-ventilated room and was fed a well-balanced ration. At this time, the bull

*Received for publication, June 20, 1934.
†Deceased, May 4, 1934.

was treated with a copper sulfate solution for the removal of internal parasites, after a fecal examination had shown a moderately heavy infestation. Under these conditions, a slight improvement was noted in the general physical condition and some of the growths sloughed off.

Attempts made to remove the warts by applying 5 per cent formaldehyde in aqueous solution proved to be unsucccessful.

In February, 1934, the physical condition of the animal became noticeably worse. A blood examination made at this time revealed an anemia. The wart condition showed no further improvement after this first sloughing. This decline in the physi-

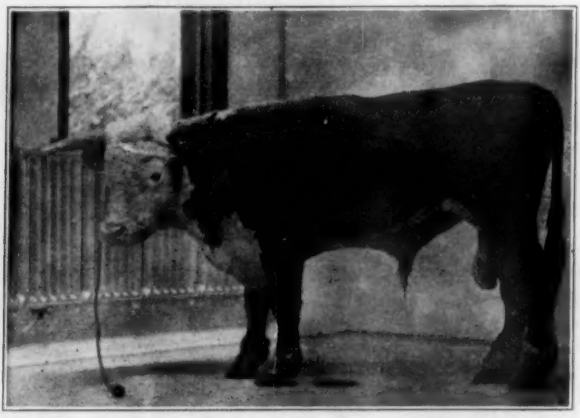

FIG. 1. Multiple papillomas on legs and scrotum of Hereford bull.

cal condition progressed until June 8, 1934, at which time he was killed and postmortem examination made.

Subcutaneous swellings noted in the cervical region proved to be pus pockets. The liver appeared to be most severely affected. Large whitish spots covered approximately half the area of this organ, which upon sectioning revealed the presence of pus. Symptoms of inflammation were noted on the gall-bladder and on the mucous membrane of the small intestine. Other internal organs appeared to be normal.

Cultures made from the heart, liver and spleen proved positive for bacteriological growths. Three types of bacteria were obtained from these cultures and isolated in pure form. A coccus type proved to be non-pathogenic for guinea pigs, whereas the two other types, which were small rods, proved fatal to guinea pigs in 17 hours after inoculation into the peritoneal cavity.

THE HISTOLOGY OF EQUINE ENCEPHALOMYELITIS. E. Weston Hurst. Jour. Exp. Med., lix (1934), 5, p. 529.

The virus of equine encephalomyelitis (eastern type) evokes in the horse, calf, sheep and dog an unusually intense encephalomyelitis, characterized by acute primary degeneration of nerve cells, the appearance in neurons of the brain system and elsewhere of nuclear inclusions resembling those in Borna disease and poliomyelitis, polymorphonuclear infiltration in the nervous tissues, with early microglial proliferation and perivascular cuffing, with mononuclears and polymorphonuclears in varying proportions. The gray matter is affected more than the white. Lesions may be less marked in the striatum, brain stem and cord than in the cerebral cortex, thalamus and hypothalamic region, and are always of low grade in the cerebellum. Meningeal infiltration is secondary. Similar changes produced in the horse by the western strain of virus are less intense and extensive. In the guinea pig, rabbit and mouse, the eastern virus causes an acute encephalomyelitis which, as is usual in neurotropic virus diseases of these lowly species, has a tendency to affect the higher olfactory centers. In addition to inclusions in the nerve cells, tiny oxyphilic bodies occur with less frequency in the glial and mesodermal nuclei of the guinea pig. In this animal, too, interstitial or bronchopneumonia may complicate the picture.

LABORATORY METHODS FOR THE DETECTION OF MILK FROM COWS INFECTED WITH MASTITIS. W. V. Halversen, V. A. Cherrington and H. C. Hansen. Jour. Dairy Sci., xvii (1934), 4, p. 281.

Leucocytes appearing in milk in excess of 100,000 per cubic centimeter and catalase in sufficient quantity to produce 2.5 cc of oxygen or more are reliable indices of udder infections. Chronic or subclinical mastitis may be detected in this manner. The addition of clear fresh blood-serum from blood-cells to milk increases the catalase content markedly. The addition of serum also decreases the curd tension. Chemical tests regularly employed for the detection of pus in urine have been negative when

applied to milk known to originate from cases of subclinical mastitis.

RATE OF PASSAGE OF INERT MATERIALS THROUGH THE DIGESTIVE TRACT OF THE BOVINE. L. A. Moore and O. B. Winter. Jour. Dairy Sci., xvii (1934), 4, p. 297.

The rate of passage of iron oxide through the digestive tract was studied in three cows. The rate of passage of rubber rings was studied in six cows. The iron oxide first appeared in the feces in from 9 hours and 55 minutes to 13 hours and 20 minutes. The high point of excretion was 33 hours and 10 minutes. The rubber rings first appeared in from 10 hours and 45 minutes to 19 hours and 30 minutes. The high point of excretion was from 23 hours and 12 minutes to 60 hours and 30 minutes. It appeared that those animals under comparable conditions which first excreted the test material and reached the high point of excretion also showed the fastest rate of excretion.

STUDIES IN SURRA. I. The blood chemistry in equine trypanosomiasis *(Trypanosoma evansi)*. Raymond Randall. Philippine Jour. Sci., liii (1934), 1, p. 97.

Trypanosoma evansi causes death in equines not by the production of a toxic substance liberated by the disintegration of the organisms, nor by the exhaustion of the blood sugar and glycogen reserve, but by an asphyxia from an uncompensated acidosis, the mechanism of which is still unknown. As the animals reached the terminal stage of infection, it was noted that there was an increase in both red and white cell counts, which may be assumed to be due to a terminal dehydration of the animal.

PASSAGE OF BOVINE BRUCELLA THROUGH SWINE. H. L. Gilman, C. H. Milks and R. R. Birch. Jour. Inf. Dis., liv (1934), 2, p. 171.

An attempt was made to ascertain whether passage of bovine strains of Brucella through a series of sows would induce these strains to assume the characteristics of the porcine type, as determined by the dye-tolerance tests. Three strains of Brucella recovered from the milk of cattle and classified as bovine by the dye-tolerance test of Huddleson were used. One strain was passed through a series of two hogs, two strains were passed through two series of five hogs each, and one strain through a series of six hogs. Infection was accomplished through intravenous inoculation. The authors were unable to detect any change from the

bovine to the porcine type, as determined by the reactions of the strains to the presence of basic fuchsin or thionine in the mediums in dilution of 1:50,000.

MICROSCOPIC DEMONSTRATION OF ACID-FAST BACILLI IN TUBERCULOUS FILTRATES AND THE PRODUCTION OF TUBERCULOUS "ULTRAVIRUS" INFECTIONS IN GUINEA PIGS. Ernest Linwood Walker and Marion A. Sweeney. Jour. Inf. Dis., liv (1934), 2, p. 182.

The claims of Fontès, Vaudremer, Calmette and others that an atypical infection of guinea pigs may result from inoculation with filtrates of tuberculous material through supposedly bacteria-proof filters have been substantiated. The frequent passage of a few acid-fast bacilli through all types and grades of filters used by these investigators was demonstrated by microscopic examination of stained smears of centrifuge precipitates of the filtrates. By inoculation with tuberculous filtrates, which were proved microscopically to contain a few tubercle bacilli, a typical non-progressive infection ascribed to the "ultravirus" was produced. The frequent passage of a few tubercle bacilli through bacterial filters would account for all of the phenomena consequent to inoculation with tuberculous filtrates without invoking the existence of Calmette's tuberculous "ultravirus." The passage of tubercle bacilli through supposedly bacteria-proof filters creates presumptive evidence that other bacteria also may pass filters.

SUITABILITY OF HERROLD'S EGG YOLK-AGAR MEDIUM FOR ISOLATION OF THE BOVINE TUBERCLE BACILLUS. William H. Feldman. Jour. Inf. Dis., liv (1934), 2, p. 194.

A study was made to determine the suitability of Herrold's egg yolk-agar medium for isolation and subsequent growth of the organisms of bovine tuberculosis. Of 71 lymph-nodes obtained from tuberculin-positive cattle, 63 were eventually proved to be tuberculous by inoculation of guinea pigs. Acid-fast bacillary forms were present in 55 of the direct smears made from 70 of the specimens. Cultures were obtained from 43 (68 per cent) of the 63 specimens in which cultivable bacteria were assumed to have been present by virtue of guinea-pig tests. There was no significant enhancement of the capabilities of growth of the bovine tubercle bacillus following its passage through guinea pigs. Pointing out that all types of tubercle bacilli will not grow with equal facility on the same medium, the author concludes that

guinea pigs rather than culture mediums should be used for the laboratory diagnosis of clinical material.

BACTERIAL ACTIVITY IN DIFFERENT LEVELS OF THE INTESTINES AND IN ISOLATED SEGMENTS OF SMALL AND LARGE BOWEL IN MONKEYS AND IN DOGS. G. M. Dack and Elizabeth Petran. Jour. Inf. Dis., liv (1934), 2, p. 204.

Gastric acidity was found not to be a constant barrier to the entrance of organisms into the intestinal tract. No bactericidal substances were observed in the contents of the small intestine. Peristalsis and secretions play an important role in rapidly freeing the small intestine of organisms. The rich fecal flora in the terminal ileum is not due to a difference in the bowel at this level but represents regurgitation of cecal contents. Isolated segments of colon in two monkeys were found to contain large numbers of *B. coli* and appreciable numbers of streptococci when tested six months following the operation in one case and three months following it in the other. Apparently the colon bacillus is peculiarly adapted to grow and remain viable in the mucous secretions of the empty large bowel.

OXIDATION-REDUCTION STUDIES OF GROWTH AND DIFFERENTIATION OF SPECIES OF BRUCELLA. C. D. Tuttle and I. Forest Huddleson. Jour. Inf. Dis., liv (1934), 2, p. 259.

Electrodes made of graphite (Acheson) are suitable for measuring the oxidation-reduction reactions of aerobic bacteria in liquid medium under aerobic conditions. The reduction potentials of Brucella in beef-liver infusion broth under aerobic conditions show a negative drift that attains the E_h value + 0.15 to 0.09 volt at the end of an incubation period of eight days. *Br. suis* shows a slightly more negative drift than the two other species. *Br. abortus* in the presence of thionine is unable to reduce the potential of the medium. *Br. suis* in the presence of basic fuchsin is unable to reduce the potential of the medium. Neither thionine nor basic fuchsin retards the negative drift of the potential of the medium caused by the growth of *Br. melitensis*.

Packers say a cow has 400 by-products. Probably her moo is sold to the crooner.

12th International Veterinary Congress
New York—August 13-18, 1934

Regular Army

Orders assigning Lt. Colonel Horace S. Eakins to duty at Fort Clark, Texas, effective upon completion of his tour of foreign service are revoked.

Orders assigning Major Allen C. Wight to Carlisle Barracks are revoked, and Major Wight is relieved from assignment and duty at the Army Veterinary School, Army Medical Center, Washington, D. C., effective on or about July 5, 1934, will then proceed to Fort Benjamin Harrison, Ind., and report for duty.

Major Clifford E. Pickering is relieved from further assignment and duty with the 1st Medical Regiment, Fort Bliss, Texas, effective in time to proceed to March Field, Calif., and report to the commanding officer, not later than May 31, 1934, for duty.

Major Charles M. Cowherd is relieved from further assignment and duty at Fort Sam Houston, Texas, effective in time to enable him to proceed to Fort Hoyle, Md., and report not later than June 15, 1934, to the commanding officer for duty.

By direction of the President, the retirement of Colonel Wm. P. Hill from active service on May 31, 1934, under the requirements of the act of Congress approved June 30, 1882, and the provisions of the act of Congress approved April 23, 1930, is announced. Colonel Hill attained the age of sixty-four years on May 13, 1934.

Major Louis L. Shook is relieved from assignment and duty at the U. S. Military Academy, West Point, N. Y., effective in time to proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about July 17, 1934, for San Francisco, Calif., and upon arrival to proceed to Fort Bliss, Texas, and report for duty.

Major Harry E. Van Tuyl is relieved from duty at the Kansas State College of Agricultural and Applied Science, Manhattan, Kan., to take effect at such time as will enable him to proceed to Fort Hamilton, N. Y., and report not later than June 15, 1934, to the commanding general for duty. The name of Major Van Tuyl is removed from the detached officers' list effective upon relief from his present duty.

First Lt. John L. Owens is assigned to duty at Fort Riley, Kan., effective upon completion of his present tour of foreign service in Panama.

Orders directing Colonel James R. Shand and Lt. Colonel Daniel B. Leininger to sail on transport scheduled to leave San Francisco, Calif., on or about July 24, 1934, for New York, N. Y., and upon arrival in New York to proceed to Washington, D. C., and report to the commanding officer, Army Medical Center, for duty, are revoked.

Major Oscar C. Schwalm is relieved from further assignment and duty with the 1st Cavalry Division, Fort Bliss, Texas, effective in time to proceed to San Francisco, Calif., and sail on transport scheduled to leave that port for New York on or about July 24, 1934; upon arrival in New York will proceed to Washington, D. C., and report to the commanding officer, Army Medical Center, for duty for the purpose of pursuing a course of instruction at the Army Veterinary School.

Orders assigning 2d. Lt. Wayne O. Kester to the General Dispensary, U. S. Army, and additional duty as attending veterinarian at Army War College and Bolling Field, D. C., upon completion of his course of instruction at Medical Field Service School, Carlisle Barracks, Pa., are revoked, and Lt. Kester will proceed to Fort Bliss, Texas, for duty instead.

Major Chauncey E. Cook is relieved from further assignment and duty at Fort Leavenworth, Kan., effective in time for him to proceed to Washington, D. C., and report to the commanding officer, General Dispensary, U. S. Army, not later than June 20, 1934, for duty and in addition will act as attending veterinarian at the Army War College and Bolling Field, D. C.

Veterinary Reserve Corps

New Acceptances

Conrad, Burton Wesley..Capt.....620 Main St., Sabetha, Kan.
Bunton, Samuel Emitt, Jr..2nd Lt...Del Rio, Tex.
Jones, Wm. Laurie.....2nd Lt...612 Delaware St., Leavenworth, Kan.
Kissileff, Alfred.....2nd Lt...Bethlehem Pike, Flourtown, Pa.

Promotions

To

Selby, Orval Cogswell.....Lt. Col...104 So. 4th St., Mankato, Minn.
Shoaff, Walter Powell....Capt.....217 Buena Vista, Paris, Ill.

Transferred to Aux.-Res.

Spencer, Hume Francis...Capt.....2928 Paseo Del Refugio, Santa Barbara, Calif.

Tuberculosis Eradication Proceeds in Minnesota

A communication from Dr. Chas. E. Cotton, of Saint Paul, Minn., secretary and executive officer of the Minnesota State Live Stock Sanitary Board, discloses the fact that that body will be in a position to designate all of the counties in Minnesota as modified accredited tuberculosis-free areas by September, 1935. All of the cattle in the 87 counties of Minnesota have been tuberculin-tested under the area plan of control, as provided by the State law. At the present time, 72 counties are officially designated as modified accredited tuberculosis-free areas by the State Live Stock Sanitary Board and the Bureau of Animal Industry, U. S. Department of Agriculture. The Board will continue to make retests of all infected herds, until all have passed complete negative tests.

12th International Veterinary Congress
New York—August 13-18, 1934

TWELFTH INTERNATIONAL VETERINARY CONGRESS

Waldorf-Astoria Hotel, New York, N. Y.
August 13-18, 1934

OFFICERS

Chairman of the Organizing Committee: Dr. A. Eichhorn.
Vice-Chairman: Dr. L. A. Merillat.
Treasurer: Dr. John R. Mohler.
General Secretary (to whom all communications should be addressed):
Dr. H. Preston Hoskins, 221 N. La Salle St., Chicago, Ill.

Membership Campaign

New York continues to lead the membership list of the states, according to figures compiled up to June 23. Four other states, California, Ohio, New Jersey and Connecticut, occupy the same relative positions that they held last month. Pennsylvania has annexed enough new members to appear third in the list, and Missouri has moved up from ninth to sixth place. Illinois has dropped from third place to fifth, and Colorado now occupies eleventh position, having stepped back from eighth. All of the 20 leaders increased their membership showing considerably, which is shown by the following figures:

New York	184	Colorado	40
California	114	Oklahoma	31
Pennsylvania	87	Wisconsin	31
Ohio	70	Connecticut	27
Illinois	69	Iowa	25
Missouri	58	Kansas	23
Minnesota	57	Indiana	22
District of Columbia	47	Maryland	22
Massachusetts	45	New Hampshire	22
New Jersey	43	Michigan	19

Chile appears on the list of foreign countries for the first time, and sends in enough memberships to rank third, surpassed only by Germany and Great Britain. Other countries that make their first appearance this month are France, The Netherlands, Dutch East Indies and Australia, and all make a splendid showing. The 14 leaders in this group are as follows:

Germany	124	Dutch East Indies	13
Great Britain	66	Egypt	13
Chile	27	Japan	1[illegible]
France	24	Australia	10
Canada	17	Union of South Africa	9
Netherlands	16	Sweden	8
Spain	15	Switzerland	7

In addition, 25 other countries are represented in the membership showing, a gain of seven new countries within the past month. The United States is going to have to look to its laurels in the competition!

Finances

With the exception of a contribution of $2 from the Army Veterinary School, which brings the total of that group up to $81, no contributions were received from veterinary organizations during the past month. The grand total now stands at $6,336.

Honor Roll

The honor roll is enhanced by one more name, that of Dr. J. B. Engle, of Summit, N. J., whose contribution of $100 makes the twenty-fourth that has been received up to June 23.

Congress Notes

Dr. Luis Santa María has been appointed official delegate of the Government of Mexico.

Prof. Dr. P. Rubay, who represents Belgium on the Permanent Commission, has also been named official delegate from his country to the Congress.

Dr. G. Flückiger and Sir Arnold Theiler are to do the honors for Switzerland at the Congress. Dr. Flückiger, who is the Swiss delegate on the Permanent Commission, is on the program for a report on foot-and-mouth disease.

Dr. P. J. du Toit, in addition to representing the Union of South Africa officially at the Congress, will also be the delegate from the University of Pretoria.

Capt. R. W. M. Mettam will represent the Protectorate of Uganda, in Africa.

Mr. C. J. Cooper, V. S., will attend as the official delegate from Bermuda.

Mr. H. V. M. Metivier, M. R. C. V. S., has been chosen the official representative of Trinidad.

Dr. T. K. Pan has been named to represent the National Health Administration of China.

Dr. Sven Wall and Prof. Gerhard Forssell will take part in the Congress as official delegates from Sweden.

Dr. Garcia Mata, Agricultural Attaché of the Argentine Embassy in Washington, D. C., has been designated as "observer" at the Congress by the government of Argentina.

Dr. F. B. Hadley, chairman of the Department of Veterinary Science in the College of Agriculture, University of Wisconsin, Madison, has been designated official delegate by the College.

Dr. Thomas W. M. Cameron is to represent the Senatus Academicus of Edinburgh University, Edinburgh, Scotland, as well as the Institute of Parasitology of McGill University, Montreal, Quebec, as previously reported in the JOURNAL.

Prof. Oluf Bang has been named official representative of the Royal Veterinary and Agricultural College of Copenhagen, Denmark.

Prof. G. H. Wooldridge, of the Royal Veterinary College of London, is to represent the Central Division, and Capt. J. R. Barker, the Midwest and South Wales Division, of the National Veterinary Medical Association of Great Britain and Ireland.

Mr. Elias Fernandini has been designated the delegate of the Association of Peruvian Live Stock Breeders, Lima, Peru.

Dr. W. Horner Andrews will represent the Agricultural Research Council, of London, England.

Capt. L. S. Balls will attend as the delegate of the Veterinary Practitioners' League of Great Britain.

June 22, 1934, was another banner day in the office of the General Secretary of the Congress. On that day, the number of Ordinary Members enrolled for the Twelfth International Veterinary Congress reached 1546, which was the total number of Ordinary Members enrolled for the Eleventh Congress held in London in 1930. And the London Congress set the record for all previous Congresses! So, in view of these facts, and with the membership list being increased daily, the 1934 Congress looks like a record-beater on all counts.

As announced elsewhere, the German delegation of 29 members is to arrive in New York on August 10. Two days later, the British party will touch American soil at Boston, traveling by rail to New York.

Eight pages of the *Veterinary Record*, for May 5, 1934, are devoted to the Twelfth International Veterinary Congress, with pictures, descriptive articles, itineraries and an excellent editorial urging British veterinarians to support the Congress in all ways. This special Congress number of the *Record* gives complete information on traveling accommodations, and carries a full-page photograph of the Waldorf-Astoria Hotel. It should make a valuable handbook for the British veterinarians who plan to attend the Congress.

Prof. Stang Heads German Delegation

Dr. Valentin Stang, professor and director of the Institute for Animal Husbandry, of the Veterinary College of Berlin, will head the German delegation to the Congress, which, according to the most recent information, consists of 29 veterinarians. The party will arrive in New York on August 10. Prof. Stang is prominent in Congress affairs, being a member and assistant secretary of the Permanent Commission, the official representative of Germany, and a contributor to the program on "Principles of Scientific Feeding."

PROF. VALENTIN STANG

Prof. Stang was born at Stuttgart, Germany, May 22, 1876, and was educated at the Veterinary College of Berlin. He did postgraduate work at the Universities of Freiburg and Strasbourg. In 1903, he entered the Ministry of Agriculture of Alsace-Lorraine and, from 1909 to 1919, he served as Director of the Institute of Animal Husbandry of that province. In 1919, he returned to Berlin to become connected with the Meat Inspection Institute of that city, serving there until 1923, when he became Professor of the Veterinary College of Berlin and a director in the Ministry of Agriculture. From 1928 to 1930, he served as Rector of the Veterinary College.

PROGRAM

TWELFTH INTERNATIONAL VETERINARY CONGRESS

Waldorf-Astoria
New York, N. Y.

Monday Morning, August 13, 10:30 A. M.

Grand Ballroom (Third floor)

OPENING SESSION

Agenda:

Call to Order by the Chairman of the Organizing Committee.
Opening Address.
Organization of the Congress.
Address of the President of the Congress.
Election of Honorary Members.
Report of the General Secretary.
Greetings from official delegates of foreign countries.
Announcements.

Monday Afternoon, August 13, 2 P. M.

SECTIONAL MEETINGS

Section I. Pathology, Bacteriology and Contagious Diseases.

East Foyer (Third floor)

ANTHRAX (NEW METHODS OF PROTECTIVE INOCULATION).

Reporters:

M. LE DR. MARIO MAZZUCCHI, Milan, Italy.

JOSEP VIDAL MUNNÉ, Jefe del Departamento Veterinario del Instituto de Investigaciones Sanitarias, Barcelona, Spain.

M. LE PROFESSEUR P. RIEGLER, Directeur de l'Institut Pasteur de la Faculté de Médecine vétérinaire, Bucharest, Roumania.

PROFESSOR DR. ALCIDES GODOY, Institute Oswaldo Cruz, Rio de Janeiro, Brazil.

Note: The time shown in this program is Eastern Daylight Saving Time, which is one hour faster than Eastern Standard Time.

Monday Afternoon, August 13, 2 P. M.

LYMPHADENITIS OF SHEEP.

Reporter:

PROFESOR DR. ANTONIO CASSAMAGNAGHI, Decano de la Facultad de Veterinaria de Montevideo y Director del Instituto de Bacteriología de la misma, Montevideo, Uruguay.

MOSQUITOES AS VECTORS OF THE VIRUS OF EQUINE ENCEPHALOMYELITIS.

Reporter:

LT. COL. R. A. KELSER, Veterinary Corps, United States Army, Corps Area Veterinarian, First Corps Area, Boston, Mass., U. S. A.

Section II. Medicine, Surgery and Obstetrics.

Grand Ballroom (Third floor)

INFECTIOUS MASTITIS.

Reporters:

PROFESSOR DR. W. STECK, Director der Veterinärmedizinischen Klinik der Universität Bern, Berne, Switzerland.

F. C. MINETT, M.B.E., D.Sc., M.R.C.V.S., Director of the Research Institute in Animal Pathology, Royal Veterinary College, London, England

PROFESSOR DR. SVEN WALL, Director of the Swedish State Veterinary Bacteriological Institute, Stockholm, Sweden.

PROFESSOR M. CHRISTIANSEN and PROFESSOR FOLMER NIELSEN, Royal Veterinary and Agricultural College, Copenhagen, Denmark.

DR. F. S. JONES and DR. RALPH B. LITTLE, Department of Animal and Plant Pathology, Rockefeller Institute for Medical Research, Princeton, N. J., U. S. A.

Section IV. Fowl Diseases.

West Foyer (Third floor)

FOWL-POX.

Reporters:

CAPT. T. M. DOYLE, F. R. C. V. S., D. V. S. M., Research Officer, Veterinary Laboratory, Ministry of Agriculture and Fisheries, Weybridge, England.

DR. W. T. JOHNSON, Poultry Pathologist, Department of Veterinary Medicine, School of Agriculture and Experiment Station, Oregon State Agricultural College, Corvallis, Ore., U. S. A.

Monday Evening, August 13, 8:30 P. M.

Grand Ballroom (Third floor)

Reception to Delegates and Members of the Congress by the Organizing Committee. Dancing will follow.

Tuesday Morning, August 14, 9 A. M.

Grand Ballroom (Third floor)

GENERAL SESSION

I. (a) New Plans for the Combating of Enzoötic Diseases Under a State Veterinary Service.

Reporter:

M. LE PROFESSEUR DR. E. LECLAINCHE, Directeur de l'Office international des Épizooties, Paris, France.

I. (b) Relationship of Veterinary Science to Animal Breeding and Public Health. Legal Protection of the Practice of Veterinary Science.

Reporter:

DR. JOHN R. MOHLER, Chief of Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C., U. S. A.

Tuesday Afternoon, August 14, 2 P. M.

SECTIONAL MEETINGS

Section II. Medicine, Surgery and Obstetrics.

Grand Ballroom (Third floor)

DISEASES OF YOUNG ANIMALS.

Reporters:

W. L. WILLIAMS, Professor of Obstetrics and Research Professor in the Diseases of Breeding Cattle, Emeritus, New York State Veterinary College, Cornell University, Ithaca, N. Y., U. S. A.

PROFESSOR DR. H. MIESSNER, Direktor des Hygienischen Institutes der Tierärztlichen Hochschule, Hanover, Germany.

PROFESSOR DR. JAN SIGMUND, Direktor des Tierarznei-Institutes der Hochschule für Bodenkultur, Prague, Czechoslovakia.

Section III. Veterinary Parasitology and Parasitic Diseases.

Perroquet Suite (Fourth floor)

COCCIDIOSIS.

Reporters:

M. LE DR. C. H. PÉRARD, de l'Institut Pasteur, Paris, France.

PROFESSOR DR. LAURO TRAVASSOS, High School of Agriculture and Veterinary Medicine, Rio de Janeiro, Brazil.

Section IV. Fowl Diseases.

West Foyer (Third floor)

CORYZA.

Reporters:

DR. J. R. BEACH, Professor of Veterinary Science, Division of Veterinary Science, College of Agriculture, University of California, Berkeley, Calif., U. S. A.

PROFESSOR DR. L. DE BLIECK, Direktor des Institutes für Parasitäre und Infektionskrankheiten der Reichsuniversität zu Utrecht, Utrecht, Netherlands.

Tuesday Afternoon, August 14, 2 P. M.

PSITTACOSIS.

Reporter:

PROFESSOR DR. PHIL. KARL F. MEYER, Director of the George Williams Hooper Foundation for Medical Research, University of California, San Francisco, Calif., U. S. A.

Section V. Tropical Diseases.

East Foyer (Third floor)

THE PIROPLASMOSES (CLASSIFICATION).

Reporters:

M. LE PROFESSEUR DR. W. L. YAKIMOFF, Directeur du Laboratoire de Parasitologie de l'Institut vétérinaire à Léningrade, Léningrade, U. S. S. R.

H. E. HORNBY, O. B. E., F. R. C. V. S., D. V. S. M., Director of Veterinary Services and Animal Husbandry, Mpwapwa, Tanganyika Territory, East Africa.

SPIROCHETOSIS.

Reporter:

PROFESSOR DR. A. KLARENBEEK, Tierärztliche Fakultät der Reichsuniversität zu Utrecht, Utrecht, Netherlands.

AFRICAN HORSE SICKNESS.

Reporter:

DR. OTTO NIESCHULZ, Tropenabteilung des Institutes für Parasitäre und Infektionskrankheiten der Reichsuniversität zu Utrecht, Utrecht, Netherlands.

ANAPLASMOSIS.

Reporter:

P. J. DU TOIT, B. A., Dr. Phil., Dr. Med. Vet., D. Sc., Director of Veterinary Services and Animal Industry, Department of Agriculture, Onderstepoort, Pretoria, Union of South Africa.

Tuesday Evening, August 14, 8 P. M.

Grand Ballroom (Third floor)

Opening Session of 71st Annual Convention of the American Veterinary Medical Association.

Wednesday Morning, August 15, 9 A. M.

GENERAL SESSION OF SECTION I

Section I. Pathology, Bacteriology and Contagious Diseases.

Grand Ballroom (Third floor)

TUBERCULOSIS (ERADICATION, IMMUNITY AND PROTECTIVE INOCULATION).

Reporters:

DR. E. A. WATSON, Chief Pathologist, Animal Diseases Research Institute, Health of Animals Branch, Department of Agriculture, Hull, Quebec, Canada.

Wednesday Morning, August 15, 9 A. M.

DR. T. VAN HEELSBERGEN, Abteilungsvorsteher am Zentral-Laboratorium für Hygiene und Volksgesundheit, Utrecht, Netherlands.

M. LE DR. C. GUÉRIN, Vétérinaire chef de Service à l'Institut Pasteur, Paris, France.

DR. A. E. WIGHT, Chief of Tuberculosis Eradication Division, Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C., U. S. A.

PROFESSOR DR. W. ZWICK, Direktor des Veterinärhygienischen und Tierseuchen-Institutes der Universität, Giessen, Germany.

PROFESSOR J. BASIL BUXTON, M. A., F. R. C. V. S., D. V. H., Director of the Institute of Animal Pathology, University of Cambridge, Cambridge, England.

HOG CHOLERA (SWINE FEVER), (ACTIVE IMMUNIZATION).

Reporters:

DR. M. DORSET, Chief of Biochemic Division, Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C., U. S. A.

DR. JOSEF MICHALKA, Bundesanstalt für Tierseuchenbekämpffung, Mödling near Vienna, Austria.

ACTIVE IMMUNIZATION OF DOMESTIC ANIMALS AGAINST TETANUS.

Reporter:

M. LE DR. G. RAMON, Chef de Service à l'Institut Pasteur, Garches, France.

Wednesday Afternoon, August 15, 2 P. M.

SECTIONAL MEETINGS

Section II. Medicine, Surgery and Obstetrics.

Grand Ballroom (Third floor)

PARTURIENT PARALYSIS (MILK FEVER).

Reporters:

PROFESSOR DR. R. GÖTZE, Direktor der Klinik für Geburtshilfe und Rinderkrankheiten der Tierärztlichen Hochschule, Hanover, Germany.

PROFESSOR DR. J. WESTER, Direktor der Klinik für Innere Krankheiten der Tierärztlichen Fakultät der Reichsuniversität zu Utrecht, Utrecht, Netherlands.

STERILITY.

Reporters:

PROFESSOR J. QUINLAN, F. R. C. V. S., Dr. Med. Vet., D. V. Sc., Sub-Director of Veterinary Services, Department of Agriculture, Onderstepoort, Pretoria, Union of South Africa.

Wednesday Afternoon, August 15, 2 P. M.

PROFESSOR DR. W. FREI, Direktor des Veterinärpathologischen Institutes der Universität Zürich, Zurich, Switzerland.

Dr. W. L. BOYD, Professor of Veterinary Medicine, Division of Veterinary Medicine, Department of Agriculture, University of Minnesota, University Farm, Saint Paul, Minn., U. S. A.

PROFESSOR DR. F. SCHÖTTLER, Direktor der Ambulatorischen und Geburtshilflichen Klinik der Tierärztlichen Hochschule, Berlin, Germany.

Section III. Veterinary Parasitology and Parasitic Diseases.

East Foyer (Third floor)

THERAPEUTICS OF WORM DISEASE.

Reporters:

DR. M. C. HALL, Chief of Zoölogical Division, Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C., U. S. A.

PROFESSOR K. SKRJABIN, Director of the Helminthological Institute, Moscow, U. S. S. R.

IMMUNITY AGAINST PARASITES.

Reporters:

THOMAS W. M. CAMERON, T. D., M. A., Ph. D., D. Sc., M. R. C. V. S., Research Professor of Animal Parasitology and Director of the Institute of Parasitology, McGill University, Montreal, Canada.

PROFESSOR DR. A. KOTLÁN, Kgl. Ung. Tierärztliche Hochschule, Budapest, Hungary.

Section VII. Animal Breeding and Dietetics.

West Foyer (Third floor)

GENETICS.

Reporters:

M. LE DR. G. K. CONSTANTINESCO, Professeur de Zootechnie à la Faculté de Médecine vétérinaire et Directeur de l'Institut national zootechnique, Bucharest, Roumania.

M. ETIENNE LETARD, Professeur de Zootechnie et d'Économie rurale à l'École nationale vétérinaire d'Alfort, Alfort (Seine), France.

ÁLVARO ARCINIEGA, Director del Servicio Pecuario de la Excma. Diputación de Vizcaya, Bilbao, Spain.

THE BLOOD-GROUP QUESTION.

Reporters:

M. LE DR. Z. SZYMANOWSKI, Professeur à la Faculté de Médecine vétérinaire de Varsovie, Warsaw, Poland.

PROFESSOR DR. S. SCHERMER, Direktor des Tierärztlichen Institutes der Universität, Göttingen, Germany.

M. LE DR. THOMOFF, Sofia, Bulgaria.

Wednesday Afternoon, August 15, 4 P. M.

Perroquet Suite (Fourth floor)

Meeting of House of Representatives, American Veterinary Medical Association.

Wednesday Evening, August 15, 7:30 P. M.

Sert Room (First floor)

Banquet for Government Delegates.

Thursday Morning, August 16, 9 A. M.

Grand Ballroom (Third floor)

GENERAL SESSION

II. (a) Veterinary Control of the Marketing of Milk.

Reporter:

PROFESSOR DR. R. VON OSTERTAG, Ministerialdirektor i. R., Tübingen, Germany.

II. (b) New Researches on Filtrable Viruses.

Reporters:

DR. R. MANNINGER, Professor an der Kgl. Ung. Tierärztlichen Hochschule und Direktor des Staatlichen Veterinärhygienischen Institutes, Budapest, Hungary.

HOFRAT DR. F. GERLACH, Direktor der Bundesanstalt für Tierseuchenbekämpfung, Mödling near Vienna, Austria.

II. (c) New Researches on Contagious Abortion (Bang's Disease).

Reporters:

PROFESSOR OLUF BANG, Royal Veterinary and Agricultural College, Copenhagen, Denmark.

DR. W. E. COTTON, Superintendent of Experiment Station, Bureau of Animal Industry, United States Department of Agriculture, Bethesda, Md., U. S. A.

PROFESSOR GUIDO FINZI, Faculty of Veterinary Medicine, Royal University of Milan, Milan, Italy.

GENERAL SESSION OF SECTION I

Section I. Pathology, Bacteriology and Contagious Diseases.

FOOT-AND-MOUTH-DISEASE (SPECIFIC PREVENTIVE MEASURES).

Reporters:

DR. J. TRAUM, Professor of Veterinary Science, Division of Veterinary Science, College of Agriculture, University of California, Berkeley, Calif., U. S. A.

M. LE DR. G. FLÜCKIGER, Directeur de l'Office vétérinaire du Département fédéral de l'Économie publique, Berne, Switzerland.

Thursday Morning, August 16, 9 A. M.

W. Horner Andrews, D. Sc., M. R. C. V. S., Director of Veterinary Laboratory, Ministry of Agriculture and Fisheries, Weybridge, England.

Professor Dr. O. Waldmann. Direktor der Staatlichen Forschungsanstalten auf Insel Riems, Greifswald, Germany.

Thursday Afternoon, August 16, 2 P. M.

Sectional Meetings

Section I. Pathology, Bacteriology and Contagious Diseases.

West Foyer (Third floor)

Classification of the Paratyphoid Diseases.

Reporters:

Dr. A. Clarenburg, Bacteriologist, Central Laboratory of Public Health, Utrecht, Netherlands.

Dr. Chas. Murray, Professor and Head of Veterinary Research Institute, Iowa State College, Ames, Iowa, U. S. A.

Dr. R. Standfuss, Veterinärrat, Leiter des Staatlichen Veterinär-Untersuchungsamtes, Potsdam, Germany.

M. Jean Verge, Professeur à l'École nationale vétérinaire d'Alfort, Alfort (Seine), France.

Section VI. Hygiene of Meat and Milk.

Grand Ballroom (Third floor)

Pastuerization of Milk.

Reporter:

Dr. H. Zeller, Oberregierungsrat im Reichsgesundheitsamt, Veterinärabteilung, Berlin-Dahlem, Germany.

Unification of the Methods of Meat Inspection.

Reporters:

M. le Professeur Dr. H. C. L. E. Berger, Directeur du Service vétérinaire et Inspecteur en chef vétérinaire de l'Hygiène publique des Pays-bas, The Hague, Netherlands.

Lt. Col. T. Dunlop Young, O. B. E., D. V. S. M., M. R. C. V. S., F. R. S. I., London, England.

Dr. E. C. Joss, Assistant Chief of Meat Inspection Division, Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C., U. S. A.

Section VII. Animal Breeding and Dietetics.

East Foyer (Third floor)

Deficiency Diseases.

Reporters:

Professor Dr. J. Marek und Professor Dr. O. Wellmann, Kgl. Ung. Tierärztliche Hochschule, Budapest, Hungary.

Thursday Afternoon, August 16, 2 P. M.

DR. GEORGE H. HART, Professor of Animal Husbandry and Head of Division of Animal Husbandry, College of Agriculture, University of California, Davis, Calif., U. S. A.

SCIENTIFIC PRINCIPLES OF FEEDING.

Reporters:

R. G. LINTON, Ph. D., M. R. C. V. S., Professor of Hygiene and Dietetics, Department of Hygiene, Royal (Dick) Veterinary College, Edinburgh, Scotland.

PROFESSOR DR. V. STANG, Direktor des Institutes für Tierzucht der Tierärztlichen Hochschule, Berlin, Germany.

Thursday Afternoon, August 16, 4 P. M.

Sert Room (First floor)

Closing Business Session of American Veterinary Medical Association.

Thursday Evening, August 16, 7 P. M.

Grand Ballroom (Third floor)

Congress Banquet.

Friday Morning, August 17, 9 A. M.

SECTIONAL MEETINGS

Section I. Pathology, Bacteriology and Contagious Diseases.

West Foyer (Third floor)

GAS EDEMA DISEASES (ETIOLOGY, CLASSIFICATION AND PROPHYLAXIS).

Reporters:

DR. J. P. SCOTT, Associate Professor of Pathology, Department of Pathology, Division of Veterinary Medicine, Kansas State College, Manhattan, Kan., U. S. A.

DR. F. C. KRANEVELD, Bakteriologe am Tierärztlichen Staatsinstitut, Buitenzorg, Java, Dutch East Indies.

M. LE DR. M. MIHAILESCO, Chef de travaux à la Faculté de Médecine vétérinaire, Bucharest, Roumania.

INFECTIOUS ANEMIA OF HORSES.

Reporters:

DR. JOHANNES VON MÓCSY, Privatdozent an der Kgl. Ung. Tierärztlichen Hochschule, Budapest, Hungary.

M. LE PROFESSOR DR. FR. KRÁL, Chef de la Clinique intérieure de l'École supérieure vétérinaire de Brno, Brno, Czechoslovakia.

Friday Morning, August 17, 9 A. M.

Section II. Medicine, Surgery and Obstetrics.

East Foyer (Third floor)

RECENT PROGRESS IN VETERINARY SURGERY.

Reporters:

DR. ALOIS POMMER, Privatdozent für Interne Medizin und Veterinärröntgenologie; Assistent der Medizinischen Klinik der Tierärztlichen Hochschule, Vienna, Austria.

DR. W. F. GUARD, Professor of Veterinary Surgery and Director of Clinics, College of Veterinary Medicine, Ohio State University, Columbus, Ohio, U. S. A.

GERH. FORSSELL, Professor of Surgery, Royal Veterinary College, Stockholm, Sweden.

SIR FREDERICK HOBDAY, C. M. G., F. R. C. V. S., F. R. S. E., Principal and Professor of Surgery, Royal Veterinary College, London, England.

Section IV. Fowl Diseases.

Grand Ballroom (Third floor)

PULLORUM DISEASE (BACILLARY WHITE DIARRHEA).

Reporters:

DR. H. VAN ROEKEL, Department of Veterinary Science, Massachusetts Agricultural Experiment Station, Amherst, Mass., U. S. A.

DR. K. WAGENER, Leiter des Staatlichen Veterinär-Untersuchungsamtes, Landsberg (Warthe), Germany.

FOWL PLAGUE.

Reporters:

M. LE DR. E. LEYNEN, Directeur honoraire du Laboratoire de l'Inspection vétérinaire de l'État, Brussels, Belgium.

DR. A. MOHAMMED EL KERDANY BEY, Director of the Veterinary Pathological Laboratory, Giza, Egypt.

LEUKEMIA.

Reporters:

PROFESSOR DR. KARL JÁRMAI, Institut für Pathologische Anatomie der Kgl. Ung. Tierärztlichen Hochschule, Budapest, Hungary.

DR. E. L. STUBBS, Professor of Veterinary Pathology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa., U. S. A.

NEUROLYMPHOMATOSIS GALLINARUM.

Reporters:

DR. F. D. PATTERSON, Assistant Professor, Veterinary Research Institute, Iowa State College, Ames, Iowa, U. S. A.

PROFESSOR DR. DOBBERSTEIN, Direktor des Pathologischen Institutes der Tierärztlichen Hochschule, Berlin, Germany.

Friday Afternoon, August 17, 1:30 P. M.

Trip to Walker-Gordon Farm, Plainsboro, and Rockefeller Institute, Princeton, by automobiles.

Friday Evening, August 17, 8 P. M.

Perroquet Suite (Fourth Floor)

MEETING OF SPECIAL COMMITTEE FOR THE AWARD OF THE BUDAPEST PRIZE.

(The Committee will consist of the members of the Permanent Commission of the International Veterinary Congresses, the president and honorary presidents of the Congress, the presidents of the sections of the Congress, and a delegate from the Royal Hungarian Veterinary Medical Association.)

Saturday Morning, August 18, 9 A. M.

SURGICAL CLIPIC FOR SMALL ANIMALS.

Saturday Morning, August 18, 10 A. M.

West Foyer (Third floor)

Meeting of Permanent Commission, together with the President and General Secretary of the Congress, and the Presidents of the Sections.

Agenda:

To examine resolutions proposed and conclusions submitted by General and Sectional Meetings, and to decide whether and in what form these shall be submitted to the Closing Session of the Congress for acceptance or rejection.

Saturday Morning, August 18, 11 A. M.

Grand Ballroom (Third floor)

CLOSING SESSION

Agenda:

Consideration of resolutions of General and Sectional Meetings.

On the proposition of the Permanent Commission, to fix time and place of next Congress.

Election of members of Permanent Commission.

Appointment of Finance Committee to examine accounts.

Closing Address.

Saturday, August 18, 12 Noon

West Foyer (Third floor)

Meeting of Permanent Commission.

Agenda:

Election of officers.

COMMENCEMENTS

ALABAMA POLYTECHNIC INSTITUTE

The following 15 graduates received the degree of Doctor of Veterinary Medicine from the College of Veterinary Medicine of the Alabama Polytechnic Institute at the commencement exercises on May 29, 1934:

Omar Wesley Bridges
Augustus B. Clark
James Ira Cornwell
J. S. Dering
Bill Dorrough
William Ginn
George Sam Jones
Mitchell G. McGee
Walter D. Martin
James Henry Milligan
Hubert A. Nowlin
Bruce H. Staton
Andrew L. Thome
Russell S. Wann
Frank R. Winsor, Jr.

A. AND M. COLLEGE OF TEXAS

The following senior students in the School of Veterinary Medicine of the A. and M. College of Texas received their degrees in Veterinary Medicine on June 1, 1934:

W. Z. Burke
F. B. Rogers
H. B. Thaxton
J. W. Upchurch

KANSAS STATE COLLEGE

The seventy-first annual commencement exercises of the Kansas State College were held at Manhattan on May 31, 1934. In the Division of Veterinary Medicine, 33 candidates received the degree, Doctor of Veterinary Medicine. They are:

Cirilo Lagmay Adan
Robert Louis Anderes
Herbert Willard Avery
*Marcus Lorenzo Bergsten
Addison Blair
Marvin James Busby
Duane LeRoy Cady
*Paul Edward Chleboun
Bradbury Bedell Coale
*Forrest Oliver Cox
*Walter Edward Dicke
Bernard Eugene Foote
*Frank Donald Gomez
*John Herbert Hensley
Ray Christian Jensen
*Howard Luther Kester
Arthur Henry Knost
*Alvin Rutti McDonald
Clarence Charles Merriman
*Lloyd Jacob Michael
*Clement Lambert Miller
*Haldor Thomas Mydland
Tillman Harvey Nelson
*James Bernard Nichols
Henry John William Osterholtz
*Culver Willis Rippetoe
Albert Arthur Roby, Jr.
*Carl William Schulz
*Herbert Franklin Sibert
Louise Sklar
*Arthur Rheinhart Thiele
Carl Edward Wendell
Abram Dwight Woodruff

Sixteen of the graduating class (indicated by *) received commissions as second lieutenants in the Officers' Reserve Corps of the United States Army.

Bradbury Bedell Coale was awarded high honors, and Robert Louis Anderes and Carl William Schulz were awarded honors in the Division of Veterinary Medicine.

Dr. Herman Farley (K. S. C. '26), of Manhattan, received the degree of Master of Science. His major subject was Pathology, and the title of his thesis was "Studies on Shipping Fever."

COLORADO AGRICULTURAL COLLEGE

The 1934 commencement exercises of the Colorado Agricultural College were held on June 7. The occasion was notable, since it marked the fiftieth anniversary of the first graduating class of the College. One of the members of this class was Dr. George H. Glover, now retiring as Dean of the Veterinary Division. All three members of the original class were back for the commencement exercises.

The degree, Doctor of Veterinary Medicine, was conferred on 14 graduates as follows:

Ralph C. Conklin
James B. Corcoran
Don L. Deane
Harold Hester
Arthur G. Keithly
Virgil M. Kerr
Mark B. Lindsey
Chester J. Mikel
Robert L. McCool
Rollin R. Smith
Archie D. Tolley
M. H. Toroian
John A. Utterback
Karl H. Willers

MICHIGAN STATE COLLEGE

At the seventy-sixth annual commencement exercises of the Michigan State College, June 11, 1934, the degree, Doctor of Veterinary Medicine, was conferred on 14 members of the graduating class:

James Edwin Cook
Maurice John Court
Wendell Edson Davis
Gardner Staples Eversole
Archie Henry Frank
Charles Daniel Logsdon (*cum laude*)
Glen Louis Noble
James Webb Scales
George Ellis Taylor
Harold Ross Taylor
William Robert Teeter
Leslie Fielding Thayer
John Warren Trumble
Maurice King Walters

Maurice King Walters won the Michigan State Veterinary Medical Association Prize of $25, awarded to the senior veterinary student who makes the best record in his course.

Stephen B. Lange was awarded the Veterinary Faculty Prize of $25. This prize is given at the opening of the fall term to the veterinary sophomore who has made the best scholastic record during his freshman year.

OHIO STATE UNIVERSITY

The fifty-seventh annual commencement exercises of the Ohio State University were held on June 11, 1934. The commencement address was delivered by G. Bromley Oxnam, LL.D., Litt. D., president of DePauw University.

The College of Veterinary Medicine presented 31 candidates for the degree, Doctor of Veterinary Medicine, as follows:

Charles Barnes
Judson Harmon Binnig
Glen Cecil Brandon
Harrison William Butz
Robert Wayne Day
John Harold Dinkel
William Francis Dorgan
Sam Elmer
Lloyd C. Ferguson
John Gale
Leonard Joyce Goss
Ralph Porter Hartman
David Emanuel James
Earl Forest Johnson
James Allen Johnson
Burnell Edison Knisely
Robert Laurence Knudson
Edward Mathew Lang, Jr.
Vincent Walter LiRocchi, Jr.
Henry Harrison Mabry
Anson Glenwood Madden
John Micuda
Luther Philip Miller
Charles Clifton Pearson
Edward Chauncey Phipps
Calvin Max Rodgers
Andrew J. Sirilo
Matt Jack Skala
Kenneth William Smith
Robert Markwood Smith
Clark Alberton Weaver

The degree, Master of Science in Veterinary Medicine, was presented to the following candidates:

John Harold Knapp (O.S.U., '32)
Carroll Kirkman Mingle (O.S.U., '30)
Roy Elwyn Nichols (Ont., '33)
Kenneth Frost Smith (O.S.U., '33)

President George W. Rightmire conferred the degrees, following the formal presentation of the candidates. The diplomas were awarded by Dean Oscar V. Brumley.

STATE COLLEGE OF WASHINGTON

The annual commencement exercises of the State College of Washington were held June 11, 1934. The commencement address was delivered by Hon. Clarence D. Martin, Governor of the State of Washington, who spoke on the value of education. The roster of the graduating class numbered 12, two of whom had completed work toward their degrees in February. Those receiving the

degree, Doctor of Veterinary Medicine and Bachelor of Science, during 1934 were:

Marvin Ebur Anderson
Charles Foster Haynes
Patricia Virginia Henno
William Robert Kermen
Rod C. McCornack
John Slover McFarland
Leonard Newman
William W. Papineau
Stanley Elliot Philips
Stanley Arthur Robinette
Clarence Edward Taylor
Robert R. Weller

Two others, Fortunato P. Basilio and Theodore Rosenoff, completed their courses at the end of the summer session of 1933.

Eight of the graduates took the examination before the Washington State Veterinary Examining Board on June 12 and 13. All of the graduates have definite plans for immediate employment.

IOWA STATE COLLEGE

Commencement exercises at Iowa State College were held June 11, 1934. On that occasion, the degree of Doctor of Veterinary Medicine was conferred on 60 candidates, the largest class that has ever been graduated from the Division of Veterinary Medicine. The graduates were:

Joe E. Akin
B. Verlou Allen
*John K. Allen
Jack G. Beardsley
*Gordon W. Blake
Everett A. Chapman
Roger M. Cocking
*Wm. F. Collins
Paul D. DeLay
Joseph H. Digranes
Marvin J. Durant
Olger Embretson
Harry D. Finlayson
Adolphe O. Garlie
Harold L. Geick
Carl F. Gerbracht
Russell W. Gerry
Lloyd V. Hartle
Joseph A. Henderson
*Stanley L. Hendricks
Dirk Hessing
Harold G. Hoyt
Emory G. Hughes
Eugene Butler Ingmand
Kenneth W. Irvin
Donald L. Jones
Marion J. Jones
Herbert Kanning
Carl H. Koll
Harold M. Larson
Magnus I. Lichter
Roger P. Link
Francis A. Malloy
Walter Muench
Norman M. Nelson
Donald C. Osburn
Melvin W. Osburn
*Robert M. Parker
Samuel G. Paul
Gerhart A. Pollman
Lester Proctor
Hoyt H. Raney
Ed E. Rash
Todd P. Rothrock
Robert O. Rydell
Charles R. Smit
*Carl F. Stephan
Robert C. Stewart
Basil K. Swink
*Edward E. Thompson
Earl M. Walker
N. Murray Wardall
Ernest Wentz
Donald N. Werring
John S. Whitfield
John H. Whitlock
George A. Wiedemeier
Raymond L. Wiedemeier
Frank Wilhite
Weldon A. Winslade

One of the members of the senior class, Gerald H. Long, will receive his degree at the end of the summer session.

Seven of the graduating class (indicated by *) received commissions in the Veterinary Reserve Officers' Training Corps of the United States Army.

Weldon A. Winslade was the winner of the Geo. Judisch Prize for high scholarship. The prize consists of initiation fee and membership dues in the A. V. M. A. for five years. Winners of the G. G. Graham Prizes, awarded to outstanding students in clinical medicine, were Joe E. Akin, first, and Carl F. Stephan, second.

Graduates elected to the honor society, Gamma Sigma Delta, were: Joe E. Akin, Harold L. Geick, Roger P. Link, Gerhart A. Pollman John H. Whitlock and Weldon A. Winslade. Three seniors were chosen by Phi Kappa Phi: Roger P. Link, Edward E. Thompson and Weldon A. Winslade. A list of initiates into Phi Zeta, national honor society, was published in the June issue of the JOURNAL. Dr. L. H. Schwarte (Iowa '28) was elected to Sigma Xi, national honor society of research workers.

CORNELL UNIVERSITY

The sixty-sixth annual commencement exercises of Cornell University were held on June 18, 1934, with 44 graduates receiving the degree, Doctor of Veterinary Medicine, from the New York State Veterinary College:

Osman Babson
Franklin Edwin Bancroft
Carl George Barnes
Lloyd Delos Barnes
Walter Oliver Bauer
Edward J. J. Bigert
Huston Adrian Calldemeier
Mario Joseph Cerosaletti
Kenneth William Davis
Charles Frederick Green, Jr.
Charles Sherwood Hallett
Charles Callen Higgins
William Young Higgins
Edgar Wendell Holden
Trevor Hugh Hughes
Ray Swartley Hunsberger
Morris Monroe Johnson
Erwin Haugh Jones
Bernard James Laughlin
Ellis Pierson Leonard
Joseph John Libra
Robert Edward Lormore
John Francis McCarthy
Robert Baker McClelland
Raphael Meisels
Joseph John Merenda
Malcolm Eugene Miller
Robert August Mueller
Ralph Gordon Murch
Clifford Philemon Murray
Dorwin Herman Perella
Clarence LeRoy Ranney
Frederick Henry Richardson
Arthur Bartlett Rogers
Leigh Harris Seely
Maurice Ezra Serling
Rudolph Julius Steffen
Daniel Stevens Stevenson
John Wright Terry
Armand Eugene Trudeau
Harold Bower Walters
Malcolm Raeburn Watt
Louis Wilson
Willis Francis Witter

The following prizes were awarded for the academic year, 1933-34:

The Horace K. White Prizes (Meritorius Students):
First Prize, $100.....Osman Babson
Second Prize, $25.....Ralph Gordon Murch
The Jane Miller Prizes (Veterinary Physiology):
First Prize, $25.....Henry Rudman
Second Prize, $25.....Leslie Askew Weight
The James Gordon Bennett Prizes (Surgical Clinics):
First Prize, $25.....Huston Adrian Calldemeier
Second Prize, $25.....Bernard James Laughlin
The Anne Besse Prize of $50 (Veterinary Medicine).....
.....Ellis Pierson Leonard
The Charles Gross Bondy Prizes (Small-Animal Clinics):
First Prize, $30.....Dorwin Herman Perella
Second Prize, $20.....Robert Baker McClelland
The Merry Prizes (Anatomy):
First Prize, $30.....Henry Kriesel
Second Prize, $20.....Geo. Edward Burch

UNIVERSITY OF PENNSYLVANIA

The 178th annual commencement exercises of the University of Pennsylvania were held June 20, 1934. On that occasion, 26 graduates received the degree, Doctor of Veterinary Medicine, as follows:

Frederick William Spranklin
Bert Worman Bierer
Robley Evans *(cum laude)*
James Otis Goad *(cum laude)*
Preston LeRoy Gsell
Fred Evans Hardman
Robert Leo Hummer
William Henry Ivens, Jr. *(cum laude)*
James McCartney Johnston
Lincoln Gruber Kutsher
Israel Live
Joseph Frederick Miller
George Elwood Outwater
Frank Herbert Owens, Jr.
Martin David Pearl
Horace Doan Pritchett
Stewart Moran Ross
Henry Peter Schneider
Frederick William Spranklin
Henry Haring Stover
Robert Leonard Ticehurst
Philip Shelwyn Tubis
Edwin Ellsworth Way
Milton Werrin
Nathaniel Werrin
Charles Gustavus Ziegler

The J. B. Lippincott Prize of $100, for the highest general average in examinations during the four years, and the T. E. Munce Prize of $25, for the highest average in the courses in Animal Industry, were awarded to James Otis Goad.

The Jeannette Blair Prize of $50, for the best work done in the Small-Animal Clinic, was awarded to William Henry Ivens, Jr.

The Leonard Pearson Prize of $50, awarded to the member of the senior class who has shown that he is most capable of dignifying and advancing veterinary science in research, in practice, in education, and in its relation to civilization, was won by Robley Evans.

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Medical Society was held at Murray's Restaurant, near White Plains, May 9, 1934, with 75 members and guests in attendance.

After luncheon, the meeting was called to order by President R. H. Spaulding, of White Plains. The first speaker was Mr. J. L. Barron, Sanitary Supervisor of Westchester County, who discussed the importance of the proper physical examination of dairy cattle and the necessity for efficient veterinary supervision in the production of wholesome milk. Immediately after this address, Dr. Crittenden Ross, of the Department of Health of the City of New York, discussed the work of his department in relation to dairy inspection. He referred to the mastitis demonstrations that have been held, saying that he considered them highly important. He was in favor of the bromthymol test, but felt that its general use might be misleading.

In the discussion that followed, the matter of the proper marking and disposal of cows rejected under the dairy inspection program was brought up. It was the general feeling that some plan of this kind was advisable, and that the resale of rejected animals should be prohibited. Dr. E. R. Blamey, of New York, then addressed the Society on the subject, "Convulsions in Dogs." He mentioned the various causes and pointed out the importance of proper diagnosis and the varied treatment necessary.

At the evening session, Dr. Carter, of Columbia University, explained "The Relation of the Pharmacist to the Veterinarian." Dr. C. J. McAnulty, of Atlantic City, N. J., gave his interesting illustrated lecture on dogs. He referred to the fact that approximately $100,000,000 is invested in various activities connected with the dog industry and pointed out that the number of shows now total 200 annually. He stated that there are 72 recognized breeds of dogs and explained the various characteristics of a number of these.

At the business session, five applicants were admitted to membership. The Society voted to cancel the August meeting, to prevent any conflict with the Twelfth International Veterinary Congress and the A. V. M. A. meeting. It voted, however, to hold the November meeting at Poughkeepsie as usual.

J. G. WILLS, *Secretary.*

SEVENTH ANNUAL CONFERENCE OF WORKERS IN PULLORUM DISEASE CONTROL

The seventh annual Conference of Workers in Pullorum Disease Control was held at the Ontario Agricultural College and the Ontario Veterinary College, Guelph, Canada, May 16-19, 1934. Delegates were in attendance from fifteen states and provinces: Connecticut, Delaware, Maine, Massachusetts, Michigan, New Hampshire, New Jersey, New York, Ohio, Ottawa, Pennsylvania, Quebec, Toronto, Virginia and West Virginia.

The delegates were welcomed by Dr. C. D. McGilvray, principal of the Ontario Veterinary College, and Dr. Christie, president of the Ontario Agricultural College, and were guests of the Ontario Department of Agriculture at a complimentary dinner on the evening of the second day.

The Antigen Committee and the Committee on Uniform Terminology reported on their activities during the past year, and the following papers and addresses were presented during the sessions:

"Observations Concerning Doubtful Reactors," "Viability of S. Pullorum," and "Studies in Variation of S. Pullorum," by Dr. K. L. Bullis, of Amherst, Mass.

"Variants of S. Pullorum and Factors Affecting Dissociation," by Dr. L. F. Rettger, of New Haven, Conn.

"Rapid Whole-Blood Tests," by Dr. R. L. Conklin, of Macdonald College, Quebec, Canada.

"Doubtful Reactors and Dissociation of S. Pullorum," by Dr. H. Van Roekel, of Amherst, Mass.

"Non-Specific Reactors and Titre Fluctuation," by Dr. E. L. Brunett, of Ithaca, N. Y.

"Cross-Agglutination of S. Pullorum with Other Members of the Paratyphoid Group," and "Poultry Tumors," by Dr. Erwin Jungherr, of Storrs, Conn.

"Fowl Paralysis," and "Etiology of Ruptured Egg Yolk," by Dr. C. L. Martin, of Durham, N. H.

"Epidemic Tremors," by Dr. C. A. Bottorff, of Durham. N. H.

"Fowl Cholera," by Dr. R. Gwatkin, of Toronto, Ontario.

"Coccidiosis," by Dr. H. J. Stafseth, of East Lansing. Mich.

"Internal Parasites of Poultry," by Dr. T. W. M. Cameron, of Quebec, Canada.

"Observations on the Inhibitory Effect of Brilliant Green on the Development of S. Pullorum," by Prof. E. R. Hitchner, of Orono, Maine.

"Field and Administrative Problems," by Dr. F. N. Marcellus, of Guelph, Ontario.
"Laryngotracheitis," by Dr. C. S. Gibbs, of Amherst, Mass., and C. B. Hudson, of New Brunswick, N. J.
"Fowl-Pox," by H. R. Baker, of Dover, Del.

Correspondence relative to the 1935 Conference should be addressed to Mr. C. B. Hudson, New Jersey Agricultural Experiment Station, New Brunswick, N. J., at which place that Conference will meet. J. S. G.

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The eleventh semi-annual meeting of the State Veterinary Medical Association of Texas was held at Texas A. and M. College, June 4-6, 1934. The event was designated as the "Mark Francis Meeting," in honor of Dr. Mark Francis, dean of the School of Veterinary Medicine at the College and one of the nation's outstanding veterinarians.

The meeting proper opened on Tuesday morning, attended by 78 veterinarians and 14 ladies. The program included the President's message, given by Dr. R. L. Rhea, of San Antonio; a short business session, and technical papers and discussions by Drs. Hubert Schmidt and R. C. Dunn, of College Station; J. S. Watson, of Mexia; Ashe Lockhart, of Kansas City, Mo., and L. E. Casey, of Dallas.

Dr. T. A. Sigler, of Greencastle, Ind., was in charge of a large-animal clinic in the afternoon. Dr. Sigler is an outstanding man in the profession and his work was of great value to the members seeing and hearing him.

Speakers on the program for Wednesday morning included Drs. F. W. Wood, of Berkeley, Calif.; L. I. Lucey, of Wichita Falls; Frank Breed, of Lincoln, Nebr.; T. O. Booth, of Temple; A. T. Kinsley, of Kansas City, Mo., and T. O. Scott, of Waco. A small-animal clinic was the closing feature of the program, in charge of Drs. W. G. Brock and R. A. Self, of Dallas; and P. W. Burns and A. A. Lenert, of College Station.

Nine veterinarians were granted licenses to practice in Texas, following examinations and a meeting of the State Veterinary Medical Examining Board, June 4. Those who secured licenses by examination are: Drs. J. W. Upchurch (Tex. '34), Georgetown; W. B. Rogers (Tex. '34), Kerrville; H. B. Thaxton (Tex. '34), Cherokee; T. A. Shipley (Chi. '90), Rosendale, Mo.; C. D.

Hoover (Ind. '13), Rio Hondo. Those who obtained licenses by reciprocity are Drs. Roy Lovell (Chi. '06), Arlington, and H. C. Gale (K. C. V. C. '10), Dallas.

During the Tuesday morning session, a sketch of the life of Dr. Mark Francis, prepared by Dr. U. E. Marney, of San Antonio, was read by Dr. H. B. Thaxton, of Cherokee, who had just been graduated from A. & M. College. This paper was roundly applauded. (It appears in this issue of the JOURNAL.)

A lawn party at the residence of Dr. and Mrs. Mark Francis was attended by the members and the Auxiliary. The program was arranged by Mrs. U. E. Marney, of San Antonio, secretary-treasurer of the Auxiliary. A song composed by Mrs. Marney, and dedicated to Dr. and Mrs. Mark Francis,* was sung, led by Mrs. W. R. Sanderson, of Brownwood, all joining on the chorus. Many other features, notably readings and speeches, made it a memorable occasion. The total attendance reached 130.

D. PEARCE, *Secretary.*

OKLAHOMA VETERINARY MEDICAL ASSOCIATION

The twentieth semi-annual meeting of the Oklahoma Veterinary Medical Association was held at Fort Reno, June 11-12, 1934. A new high record of attendance was established at this meeting, in spite of discouraging economic conditions and a drouth of alarming proportions. Optimism was the keynote of the meeting, and the program reflected this splendid spirit. Lively debates on Association matters, a well-balanced program, excellent facilities and conveniences, together with an abundance of clinical material, held the interest of all, from the opening to the closing minutes of the sessions.

With two exceptions, the entire program was put on by members of the Association. The exceptions were Dr. C. P. Fitch, president of the A. V. M. A., and Dr. Horace J. Harper, agronomist, Oklahoma A. & M. College. Dr. Fitch discussed "The Control of Bang's Disease," emphasizing the following points: (1) From an economic standpoint, Bang's disease is the most important live stock disease today; (2) public sentiment is crystallizing rapidly on a demand for aggressive eradication measures; (3) the eradication of the disease will never be accomplished by the use of biologic agents. Dr. Harper, an authority on soil composition, spoke on "Mineral Contents of the Soil and Their Relation

*Published on page 75 of this issue of the OURNAL.

to Animal Nutrition," a subject with which every veterinarian should be familiar if he hopes to cope successfully with many of the deficiency disorders that are causing so much concern at present.

Other speakers were: Dr. H. W. Orr, of Stillwater, on "The Mineral Requirements of Domestic Animals and Some of the Principal Deficiency Diseases"; Dr. O. E. Robinson, of Bixby, on "Digestive Disturbances of the Horse"; Dr. W. H. Martin, of El Reno, on "The Standard Milk Ordinance in Operation"; Capt. E. E. Hodgson, U. S. Army, on "Some Observations on Horse Breeding"; Dr. C. C. Hisel, of Oklahoma City, on "The Past, Present and Future of Tuberculosis Eradication in Oklahoma," and Dr. F. Y. S. Moore, of McAlester, who presented a collection of extraordinary experiences in his long practice under the caption of "Unusual and Interesting Cases I Have Met."

A round-table discussion, led by Dr. Fitch, brought out a wide variety of questions and an instructive discussion of all of them.

Clinics, for both large and small animals, occupied the entire afternoon of the second day. Captain Hodgson, in charge of the large-animal section, was assisted by Dr. E. Peterson, of Vinita, and Dr. D. H. Ricks, of Pauls Valley. Demonstrations of bot control in horses, correcting breeding difficulties in the mare, general anesthesia by the use of chloral hydrate intravenously, ovariectomy in the heifer, and methods of surgical restraint were given. Dr. S. R. Espy, of Oklahoma City, conducted the small-animal clinic, at which a number of interesting cases were presented for diagnosis and several difficult operations performed. Both clinics were well attended.

The Ladies' Auxiliary, organized during the annual meeting in January, held a business session the first day, further perfecting its organization and outlining the functions and duties of its officers. Officers for the coming year are: President, Mrs. D. H. Ricks, Pauls Valley; first vice-president, Mrs. H. W. Ayers, Oklahoma City; second vice-president, Mrs. J. C. Cummings, Cherokee; secretary-treasurer, Mrs. J. H. Wirtz, Oklahoma City. More than forty ladies were in attendance.

A well-arranged banquet was served at the Southern Hotel, El Reno, on the evening of the first day, with Dr. O. E. Robinson acting as toastmaster. Dancing and a splendid program of musical and other appropriate entertainment provided an enjoyable evening for the more than 120 in attendance.

C. H. FAUKS, *Secretary.*

NECROLOGY

HARRY MORRIS

Dr. Harry Morris, head of the Animal Pathology Department of Louisiana State University, Baton Rouge, died on May 4, 1934, following an illness of two weeks. Death resulted from an attack of influenza and the resultant rapid development of a kidney ailment from which he had suffered for some time. He was 52 years of age.

A native of Ohio and a graduate of the Ohio State University, class of 1910, Dr. Morris, soon after graduation, accepted a position as bacteriologist at the Louisiana Agricultural Experiment Station. From that time until his death, he was connected with Louisiana State University.

Dr. Morris joined the A. V. M. A. in 1913, and served as Resident Secretary for Louisiana from 1923 to 1926, and from 1930 to 1931. He was a member of the Louisiana Veterinary Medical Association, and was elected president of that organization at the meeting held in Baton Rouge in February. He is survived by one son. Mrs. Morris (née Clara Stuart) died a few years ago.

THOMAS L. DARDIS

Dr. T. L. Dardis, of Stockton, Calif., died at the Emergency Hospital, October 22, 1933, as a result of injuries received a few hours previously when his automobile crashed into the rear end of a truck. He was responding to an emergency call when the accident occurred.

Born in Santa Rosa, Calif., in 1874, Dr. Dardis was graduated from the San Francisco Veterinary College in 1908. He had lived in Stockton for about 40 years, and had practiced there since his graduation. He was well known in state veterinary circles, and was highly esteemed in racing circles for his success in treating race horses. Dr. Dardis is survived by his widow (née Clara Pahl), one daughter and two sons.

H. W. HALEY

Dr. H. W. Haley, of Marceline, Mo., died February 18, 1934. He was a graduate of the Kansas City Veterinary College, class of 1912, and had been in practice at Marceline since his graduation.

FRANK DALEY COLES

Dr. Frank D. Coles, of Norwich, Conn., died at his home, May 14, 1934, following a heart attack.

Born in Newton, Mass., August 27, 1881, Dr. Coles spent most of his early life in Voluntown, Conn. He was an honor graduate of the Ontario Veterinary College, class of 1907. Following his graduation, he practiced in Jewett City, Conn., for three years. In 1910, he located at Norwich, where he enjoyed a large and lucrative practice until his sudden death.

Dr. Coles was a member of the Connecticut Veterinary Medical Association and of the Norwich B. P. O. E. Surviving him are his widow (née Lena May Babcock), two sisters and one brother.

E. L.

CASPER H. LARSON

Dr. C. H. Larson, of Dumas, Ark., died in a hospital in Memphis, Tenn., June 4, 1934. Death was due to septic poisoning, following an automobile acccident near Dumas, on May 30.

Born in Norway, Iowa, February 22, 1900, Dr. Larson received a high school education and then entered Iowa State College. Following his graduation, in 1923, he entered practice at Lake Park, Iowa. Later, he returned to Iowa State College as a member of the teaching staff, where he remained two years. He then operated a small-animal hospital in Miami, Fla., for several months, leaving to accept the position of Sanitary Inspector for Kansas City, Mo. After he left Kansas City, he established himself in private practice at Dumas.

Dr. Larson joined the A. V. M. A. in 1924, and was Secretary of the Arkansas State Veterinary Medical Association. Surviving are his widow (née Dorothy Morris), his parents, five brothers and one sister.

J. C. G.

S. TAYLOR YOUNG

Dr. S. Taylor Young, of Middleburg, Va., died unexpectedly June 21, 1934, at his home, from an attack of heart disease.

Born at Andrew's Bridge, Pa., August 25, 1879, Dr. Young was

a graduate of the University of Pennsylvania, class of 1903. Following his graduation, he entered general practice at Oxford, Pa. In 1916, he moved to a farm near Newark, Del., where he combined practice with farming. Later he discontinued farming to devote his entire time to his profession. Two years later, he accepted a position as veterinarian to a large horse-breeding estate near Middleburg, Va. Later he engaged in general practice at Middleburg and specialized in diseases of hunters. He also bred hunters and, during recent years, served as judge at many of the shows of horses of this class.

Dr. Young joined the A. V. M. A. in 1926. He is survived by his widow (née Margaret B. Mason), one daughter, one son, three sisters and one brother. Funeral services were held at Middleburg and the body was taken to Oxford for burial.

BERNARD B. GLOVER

Dr. Bernard B. Glover, of Lexington, Va., died on June 19, 1934, following a heart attack. He was a graduate of the Ontario Veterniary College, class of 1908, and was one of the leading practitioners of Virginia. He had served as president of the Virginia State Veterinary Medical Association. I. D. W.

Our sympathy goes out to Dr. Bassett Kirby, Woodbury, N. J., in the death of his wife on June 2, 1934.

PERSONALS

Dr. C. F. McKinney (T. H. '13), of Charleston, Ill., has located in Arthur, Ill.

Dr. Russell Wann (A. P. I. '34), of Silverwood, Ind., has decided to locate in Lexington, Ky.

Dr. F. H. Conover (Ont. '34), of Petersburg, Ill., has moved to Roseville, Ill., for general practice.

Dr. Judson Binnig (O. S. U. '34) has taken over the practice of the late Dr. J. D. Fair, at Millersburg, Ohio.

Dr. C. E. Wendell (K. S. C. '34), of Pittsburg, Kan., has opened an office at the corner of Market and Clay Streets.

Dr. Charles C. Pearson (O. S. U. '34), of Fremont, Ohio, has taken over the practice of the late Dr. F. E. Anderson, at Findlay, Ohio.

Dr. Samuel G. Dreppard (T. H. '12), of West Plains, Mo., has been laid up since February, with a broken leg, the result of an automobile accident.

Dr. C. L. Lee (Chi. '18), of Iola, Wis., will act as judge at the annual dog show of the Onwentsia Club of Chicago, at Lake Forest, Ill., July 3-4.

DR. E. C. PHIPPS (O. S. U. '34) has acquired the small-animal hospital of Dr. E. H. Queener, at Cynthiana, Ky., Dr. Queener having returned to Tennessee.

DR. H. P. HEINLEN (Gr. Rap. '13), of Niles, Mich., is the first to announce his candidacy for the Democratic nomination as county coroner in the coming primaries in Berrien County.

DR. HARVEY W. CAMPBELL (Colo. '22) has resigned his position with the California State Department of Agriculture and is now engaged in practice at 913 Santa Rosa Avenue, Santa Rosa, Calif.

DR. E. S. DEUBLER (U. P. '05), of Penshurst Farm, Narberth, Pa., was elected President of the Ayrshire Breeders' Association of America, at the recent annual meeting of that organization.

DR. C. H. READING (Mich. '27), formerly of Decatur, Mich., and for the past three years employed in the Veterinary Laboratory at the University of Wisconsin, has entered practice at Dodgeville, Wis.

DR. HASKELL LETT (Chi. '09), of Seymour, Ind., recently became associated with Dr. M. J. McGinty in operating the McGinty Dog and Cat Hospital, Inc., 829 East Washington Street, Indianapolis, Ind.

DR. B. F. RICEBARGER (Ont. '06), of Saint Charles, Ill., was kicked in the leg by a vicious horse which he was called to attend recently and was confined to his home for about a week while recovering from his injuries.

DR. R. C. KLUSSENDORF (Corn. '31), who has been associated with Drs. Downing and Lothe at Waukesha, Wis., has accepted a position in the Veterinary Control Laboratory of the Department of Live Stock Sanitation at Madison, Wis.

DR. W. T. BRINKER (O. S. U. '30), formerly veterinarian at the Cleveland Zoo, and now located at Miamisburg, Ohio, recently treated a three-year-old Brazilian ring-tailed monkey that was shipped to him in the hope of saving its life.

DR. J. H. MURPHY (T. H. '15), of the U. S. Bureau of Animal Industry, who has been stationed at Paris, Ill., for some time, recently was ordered to proceed to Bismarck, N. Dak., to assist in handling cattle stricken in that area as a result of the drouth.

DR. EDGAR H. CALLANDER (Ont. '91), of Zanesville, Ohio, has announced his candidacy for the Republican nomination for county commissioner at the primary elections to be held in August. Dr. Callander has practiced in Zanesville and Muskingum County for 41 years.

DR. HARRY H. ROSS (Ont. '05), of Brandon, Manitoba, had a bad accident on May 12, in which he received a broken nose, and his car was a total wreck. Friends of Dr. Ross will be glad to know that he has entirely recovered from the effects of the accident and that he is again able to take care of his extensive practice.

DR. E. C. JONES (K. S. C. '16), of Grand Island, Neb., recently became Vice-President of the Norden Laboratories, when the Platte Valley Serum Company, of which he was President, merged with the Laboratories. Dr. Jones will continue in charge of the plant at Grand Island, which is now known as the Norden Serum Plant.

DR. W. H. FITCH (Ont. '17), of Walcott, Iowa, was kidnaped recently by Joe Palmer, an escaped Texas gunman, and held prisoner for nearly 18 hours. He was carried to Saint Joseph, Mo., because the convict needed both a car and a driver to that city. There Dr. Fitch, with two other Iowa men who had been taken along to prevent identification of the convict, was released, but not until the desperado had robbed all three.